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THE TURN OF THE CENTURY — AND AFTER*

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BOSTON

MY talk this evening, to which I have given the above title, I will describe as a fairy tale, dealing, like most fairy tales, with events of long ago, for such it seems to me. Imagine me as a prospective student presenting myself in late September of 1897 at the door of the Harvard Medical School on the corner of Boylston and Exeter streets, Boston — a building constructed for the school fourteen years earlier and representing "the acme of convenience and suitability and believed to be adequate for at least three generations," yet already having quarters cramped for laboratory studies on account of the vast progress being made in the medical sciences. An impressive white-bearded man of responsible appearance greeted me. This was James Skillen, always known as Jim, a former male nurse or operating room orderly at the Massachusetts General Hospital, and promoted to be janitor of the medical school when it moved from the old building on North Grove Street to its new quarters. Jim lived in the school, and with his wife, Susie, and daughter, Nellie, ran it most effectively — ably assisted, of course, by the dean and faculty of the school. He knew every student and faculty member and spoke of the latter with entire respect but considerable familiarity, by their first names. Perhaps in contrast a picture should be presented of the great educator under whose direction the medical course was conducted, President Charles W. Eliot. It is impossible to do more than touch upon his influence. Mr. Eliot assumed the presidency of Harvard University in 1869, and immediately evidenced his interest in the medical school and his sense of the unity of the school with other graduate departments to form the university, by attending and presiding at the faculty meetings. It was essentially a proprietary school, with a curriculum of four months of lectures during each of two years. In addition to attendance at exactly the same lectures in succeeding years, the students were required to spend their additional eight months of each year and one succeeding year in an apprenticeship to some acceptable practitioner, or indeed in one of the very few available house-officer posts at the Massachusetts General Hospital or in the recently established Boston City Hospital. It was only necessary for the student to pass perfunctory examinations in five of the nine lecture courses in order to obtain his degree. The school had little or no endowment, and the nine professors and a few assistants were paid from the proceeds of the tickets which were sold for their lectures. Mr. Eliot increased the course to three years of graded instruction; introduced adequate written examinations; advised greater contact of students with patients, in the form especially of section teaching; and explained to Professor Henry J. Bigelow, who in faculty meeting with groans deprecated the numerous changes, that the reason they were occurring was because there was a new president. Later came the addition of the fourth year to the curriculum and the requirement of an A.B. degree or its equivalent for admission. Mr. Eliot showed a similar interest in elevating dental education to its appropriate place as a department of general medicine. He urged the systematic teaching of preventive medicine, defended free experimentation and vivisection and promoted the recognition of medical service as a part of the social organization. His contributions to medical education were incalculable.

The first teacher with whom I came in contact was Thomas Dwight, Parkman Professor of Anatomy, one of the men about whom one could say, as did a contemporaneous biographer of a colleague: "He was a legendary Bostonian, with all the crushing disadvantages of an assured position, binding tradition and a competence." Dwight graduated in medicine in 1867, and the picture of him showing a dignified, grave teacher with a full beard scarcely suggests the slender, active young man who, tradition says, while operations were

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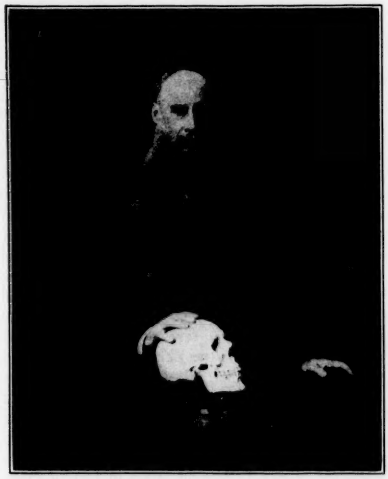
conducted in the amphitheater at the Massachusetts General Hospital under the carbolic vapor of Lister sprayed over the field from a foot-driven machine, was entrusted with a hand atomizer to promote additional efficacy, and as he bobbed up between the members of the operating group to deliver a spray over an area where it appeared to be especially needed, received many a sharp rap

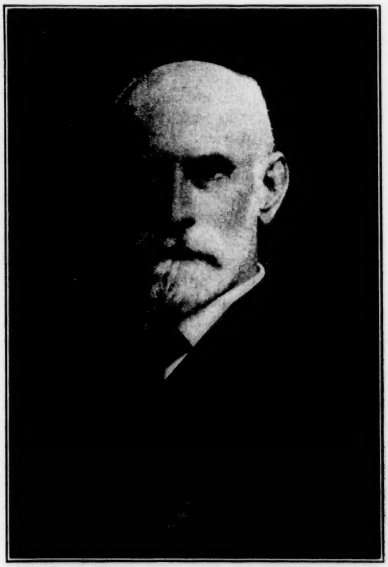


THOMAS DWIGHT

on the cranium or knuckles with the instrument in the hand of the attending surgeon. Dwight studied abroad with Rudinger at Munich, and on his return began to practice medicine and surgery, but his heart was in anatomy; he taught comparative anatomy, histology and embryology and was finally entrusted with the professorship on the retirement of Oliver Wendell Holmes. He was an effective and enthusiastic teacher; he took his duties seriously, and being a man of great faith and spirituality impressed upon his students a sense of the privilege which was accorded to them in dissecting the tenement of clay from which the soul had fled; and obtained from them the respect and decorum appropriate to the occasion. Dwight was a morphologist, and made important researches in the normal variations of the skeleton; and with Mr. Emerton, the artist and technician, he made the superb large-scale models of the bones of the skeleton which, with other means devised by him, contributed to the supreme excellence of the instruction in anatomy.

The subject of anatomy should not be closed without speaking of the demonstrator and the assistant demonstrators, a group of young surgeons, enthusiastic and interested in their work because they knew that skill in gross anatomy and dissection was the open sesame to success as surgeons, which perhaps without exception they attained. Nor should mention be omitted of Tom Bonney, the devoted and intelligent preparateur of the anatomical material, whose term of service, begun in 1892, is just now, as this is written, coming to a close.

Simultaneously with Dwight I met another great personality, Charles Sedgewick Minot, professor of embryology. Minot had the same sort of personal background as did Dwight; indeed it was actually he who was described as the "legendary Bostonian" above-mentioned. Minot was a born

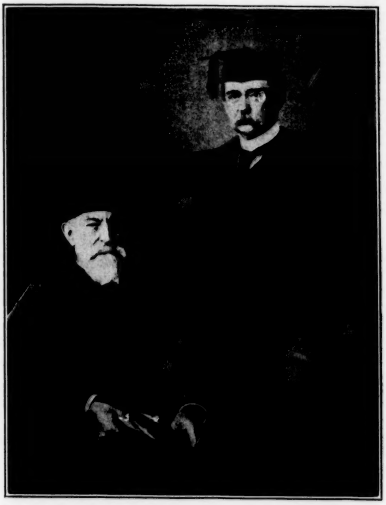


CHARLES SEDGEWICK MINOT

naturalist; he studied insects and flowers while still a schoolboy, obtained his degree in science at the Massachusetts Institute of Technology, studied with Ludwig in Leipzig and came home well trained in the biological sciences and in chemistry. No doubt he might have excelled in the latter, but he preferred morphology and mastered the technic of microscopy; he first taught embryology in 1880

and finally took charge of the Department of Histology and Embryology in 1892, with further promotion to become James Stillman Professor of Comparative Anatomy in 1905. As the scientific world knows, he made an almost unrivaled collection of the embryos of various vertebrates and perfected the microtome, superintended the staining of sections of one hundred and fifty complete series of pig embryos for study by the students, and wrote a standard treatise on embryology. I remember him as a moderately tall, very erect, well-knit figure standing behind the laboratory desk upon which rested his fingertips, describing with great deliberation, and with faultless, clean-cut enunciation, the intricacies of his subject; illustrating it also frequently with graphic diagrammatic drawings on the blackboard, of textbook perfection. He introduced us to a Dutch motto sent to him by Professor Hubrecht, of Utrecht, which was displayed in large letters and framed on the wall: *Wat baeten kaers of bril, als den uil nit sien wil?* which he translated as follows: "Of what use can light and lenses be if owlets look and will not see?" Minot believed in meticulously ascertaining and recording the student's daily work; he constructed a board looking somewhat like a cribbage board, the holes in which represented the desks of the individual students, and pegs of different colors to represent the instructors, whose personal supervision over the students was thus followed.

The course in physiology introduced me to another teacher of equal notability, Henry Pickering Bowditch, professor of physiology. As one looks at the sedate figure in cap and gown one can scarcely picture him as a brilliant cavalry officer who served throughout the Civil War in the First Massachusetts Cavalry, and who entered Richmond as a major in 1865. Bowditch was a grandson of Nathaniel Bowditch, the mathematician and navigator, and son of a successful Boston merchant; so that he also seems to be one of the legendary Bostonians who made a mark for himself in spite of handicaps of birth. Returning from the war, he graduated in medicine at Harvard in 1868, and then studied in Paris with Claude Bernard and in Leipzig with Carl Ludwig; on returning to Boston he established the first physiological laboratory for students in the United States and engaged in researches in general biology, experimental pharmacology, pathology, psychology and surgery. I remember his emphasis on productive scholarship; his inventiveness; his clear lectures and supplementary recitation periods. He introduced the plan of sending students to the original sources for material for physiological theses. He gave the impression of high breeding and was conspicuous for courtesy, fairness and good will, and sometimes failed to suppress in a classroom his sense of humor. During his long career as a teacher of physiology he actually established laboratories in three separate buildings: North Grove Street, Boylston Street, and finally in the present buildings on Longwood Avenue. Together with J. Collins Warren, he was more instrumental than any other in planning the expansion of the medical school and enlisting the interest of public-spirited men in the project. He was dean of the school from 1883 to 1893. He sought out and brought to Harvard promising men from other universities; he was a public-spirited citizen, always to be depended on to help defeat the unwise and to promote the wise in state legislation. The picture displayed of him garbed in cap and gown and seated with Warren at the time of the dedication of the new buildings in 1906 does not do justice to his expressive and interesting face, because it perhaps betrays the existence of the chronic illness which was already spreading its mask over his features.



HENRY PICKERING BOWDITCH
JOHN COLLINS WARREN

Chemistry was taught by Edward Stickney Wood, who, having graduated from Harvard College in 1867 and from Harvard Medical School in 1871 and completed a surgical internship at the Massachusetts General Hospital, studied in Berlin and Vienna and began teaching as adjunct

professor to James C. White, who was later to enter the field of dermatology. Wood became full professor in 1876; though not a genius, he was a remarkable teacher who gave the impression of perfect mastery of his subject. Interest was added to his personality by the fact that he was the most famous medical expert witness of his day and was often quoted on the front pages of the daily papers in connection with some murder trial or other medicolegal problem of absorbing interest. His students could well believe that as a medical expert witness he was without an equal — cool, calm, impartial and imperturbable under cross-examination. It is said that his advice was much relied upon by Mr. Eliot.

In pathology I was fortunate in having a teacher unusual both in ability and in personality, William Thomas Councilman, who, descended on his father's side from a settler from Holland and on his mother's side from Scotch-English forebears, was the son of a country physician and farmer in Maryland. He was a born naturalist, and while working on the farm as a boy made a collection of bones illustrating the comparative osteology of the indigenous vertebrates. After studying medicine at the University of Maryland he passed two years abroad with Chiari in Vienna, Cohnheim and Weigert in Leipzig and von Recklinghausen in Strasbourg, and on returning in 1892 joined Welch in teaching pathology at the recently founded Johns Hopkins Medical School. He was called to Harvard as the third Shattuck Professor of Pathological Anatomy, succeeding Reginald H. Fitz; and became pathologist to the Boston City Hospital, whither he attracted among others Dr. Frank B. Mallory, weaning him from clinical practice to pathology. Councilman was a man of great activity and enthusiasm in teaching and research. He promoted the appointment of a full-time pathologist at the Massachusetts General Hospital in 1896; in 1913 he became pathologist to the Peter Bent Brigham Hospital, which had just opened its doors. His was a colorful personality; as a biographer states, he "cultivated neither reserve nor mannerisms but achieved dignity by virtue of directness, competence and honesty." He inspired his students with the ambition to become independent observers and thinkers. He often acted on impulse regardless of the conventions. On one occasion at the Boston City Hospital, having just completed an autopsy he received a telephone message from the superintendent which irritated him; instantly, without coat and vest or collar and necktie, with red suspenders and underwear conspicuous, he rushed across the yard and through the buildings and passageways to "Center," where he had a talk with the astonished and probably quite reasonable McCollum. The photograph of him shows him seated looking reflectively at his dog, Pasco, lying on his desk. Pasco was the offspring of a Boston terrier belonging to Louis Pasco, familiar to more than one generation of medical students as the keeper of the morgue at the Boston City Hospital. In his last years, after his retirement, Councilman suffered from, but was not cowed by, attacks of angina pectoris, for the relief of which he always carried a bottle of nitroglycerin.

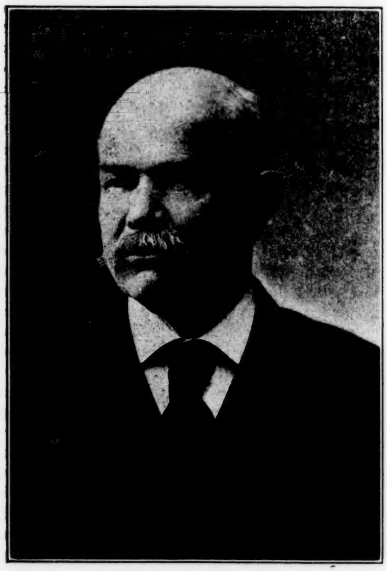
Harold Clarence Ernst, professor of bacteriology, was a pioneer in that branch. On the paternal side he was descended from a German grandfather who migrated from Prussia during the Napoleonic Wars and married a Boston lady of old New England stock. It is not frivolous to record that Ernst, who graduated from Harvard College in 1876, was one of the best amateur base-

WILLIAM THOMAS COUNCILMAN

ball pitchers of all time; he is said to have added one more to the different types of curves which could be imparted to a thrown ball, and received flattering offers to become a professional. After receiving his medical degree in 1880 he studied with Koch in Berlin, and on returning to this country gave six lectures on bacteriology in 1885 at the Harvard Medical School, probably the first lectures on that subject given in a medical school in the United States. It was hard work against opposition to establish the importance of bacteriology. Ernst developed the first sterilizing apparatus at the Massachusetts General Hospital, and for years supplied from his own little laboratory all the vaccine and antitoxin used by the City of Boston, and also tuberculin for the inoculation of cattle. He became first professor of bacteriology in 1895 and held the office until 1922. His students remember him as a tall man, large of frame, of austere and rather stern military appearance, which was much confirmed by his uniform in the World War, which gave him the appearance of a

Prussian military officer. He was an able man of strong character and great courage and devotion to high ideals, which led him to many types of public service and to the laborious editorship of the *Journal of Medical Research*. It was fortunate that the infant subject of bacteriology should have found such a forceful and competent champion.

The theory and practice of physic was taught by the Hersey Professor, Reginald Heber Fitz, who graduated from Harvard College in 1864 and from Harvard Medical School in 1868, and who was then surgical house officer at the Boston City

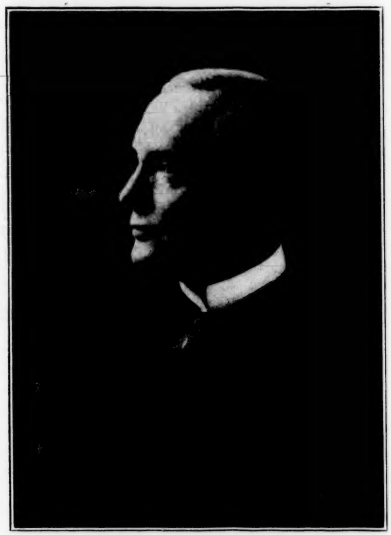


REGINALD HEBER FITZ

Hospital. Two years were then spent in Europe with Rokitansky, Oppolzer and Skoda in Vienna and Cornil in Paris, whence he returned to be one of the first to introduce into America Virchow's ideas in regard to cellular pathology. He approached clinical work through the pathological laboratory and autopsy room, succeeding John B. S. Jackson as professor of pathological anatomy, becoming visiting physician to the Massachusetts General Hospital in 1887 and finally reaching the obvious objective of Hersey Professor of the Theory and Practice of Physic in 1892. His teaching began just at the establishment of the three full years of instruction with written examinations and the requirement that every student must pass every course, and just at the beginning of laboratory teaching in physiology and chemistry. He exercised a profound influence on medical education, being chairman of the Committee on Curriculum for many years. He persuaded Henry F. Sears to give the first laboratory in America devoted exclusively to the study and teaching of pathology, housed in an addition to the building on Boylston Street. All the world knows his most conspicuous achievement: the demonstration of the responsibility of the appendix for the majority of inflammatory conditions in the right iliac fossa, brought to the attention of the world in a paper entitled "Perforating Inflammation of the Vermiform Appendix," read in 1886 before the Association of American Physicians. This paper gave the first clear picture of the pathology and clinical aspects of the disease and advocated operative interference. Three years later he urged the then startling innovation of the interval operation, and the same year published a paper on acute pancreatitis, which described fat necrosis. These are merely the most striking of his many important contributions. Fitz met the students in a series of systematic lectures and in amphitheater clinics and recitations. The lectures were remarkable as models of clear and precise exposition, rapidly delivered at a rate which made it almost impossible to take complete notes. In the clinic he required a student before the class to secure the main features of the history of a patient, make a physical examination, perform or ask information about necessary laboratory tests and then make and defend a diagnosis and suggest treatment. The ability and learning of the man and the precision which he required in observation, interpretation and exposition were not soon forgotten. He permitted the student to fall headlong into the inevitable pitfalls, whence he rescued him with humorous irony, which disconcerted the victim but delighted his fellows—a method of pedagogy which Fitz well knew was second to no other in making a lesson unforgettable. He delighted in argument and discussion and waxed more cheerful in opposition, though he was tenacious of nothing but the truth. I always felt admiration and respect, not unmixed with fear, until later when I came into more personal relations with him and was able to confirm my suspicions of friendly kindliness. A biographer says, "There must be many who owed him their best ideals in medicine."

John Collins Warren, the fourth in the Warren surgical hierarchy, great-grandson of the founder of the Harvard Medical School and grandson of one of the founders of the Massachusetts Gen-

eral Hospital, was head of the Department of Surgery. After graduation from Harvard University in arts and in medicine, he visited Lister in Glasgow, studied the antiseptic method at the hands of its creator and brought it back to America. He was particularly interested in surgical pathology and wrote an admirable textbook on that subject; and as a matter of course, he joined the surgical staff of the Massachusetts General Hospital and the faculty of the Harvard Medical School, and in 1899 was made Moseley Professor of Sur-



FREDERICK CHEEVER SHATTUCK

gery. Neither as a surgeon nor as a teacher was he endowed with great genius, but in the former capacity he was courageous, resourceful and thorough, and in the latter he gave much time to the preparation and conduct of his teaching. With the traditions of three distinguished generations pointing out his own duty and destiny, his life was a fulfillment of the adage, *Noblesse oblige,* and throughout he was a beacon light of personal distinction, charm and public usefulness. With Bowditch he conceived the magnificent project for the new school, and with his happy faculty of securing and sustaining the interest of others he had much to do with securing the necessary support; he was one of the first to advocate the plan for a dormitory. He was chairman of the Cancer Commission of Harvard University and secured the funds for the Collis P. Huntington Memorial Hospital. The respect and affection in which he was held increased with his years and were in direct proportion to a contemporary's capacity to perceive the elements of true character. The continuity of the Warren tradition is a most enviable heritage for the Harvard Medical School and for the medical profession of Boston.

The next portrait is that of the professor of clinical medicine, Frederick Cheever Shattuck, obviously another legendary Bostonian with the accompanying handicaps. He was the fourth in line of able physicians. Perhaps through his many-sided personality, his extraordinary charm and his wisdom he made as deep an impression on succeeding classes of medical students and house officers as did any other of their teachers. He had studied in London, Paris and Vienna, and brought home high standards of careful history-taking and of physical examination. Something — perhaps it was his very youthful appearance — militated against his immediate success in building up a practice, but he utilized his time to advantage with patients and students in the Out Patient Department of the Massachusetts General Hospital. The exercises he conducted were always clinical in character, and it was his actual relation with patients, his sympathetic understanding of their woes, his humorous but never satirical touch, which would best exemplify to the students the methods of the wise physician. He was astute in diagnosis and daring in his treatment. It was wisely stated that he was a successful psychotherapeutist long before that term was coined. The statement that is said to have been current in some laboratories that medicine can be practiced without seeing the patient was scornfully repudiated by him. He was fond of saying that the final test of all diagnostic and therapeutic measures is made at the bedside rather than in the laboratory. His fundamental success as a teacher gave authority to his opinion that a full-time teacher, trained only in the laboratory and hospital ward, can impart only a partial knowledge of the practice of medicine. His was a picturesque figure as he drove about the city in an open victoria, with a cigarette between his lips, a carnation in his buttonhole, a gaudy waistcoat and with reading matter — medical journals or letters — piled up on the seat beside him, and attended by Hans, the dachshund. No better influence did the medical students encounter among their preceptors than that of Shattuck. He lived through a period of enormous development in the medical sciences and their application. In these matters he was not so much a pioneer as an interpreter and practitioner.

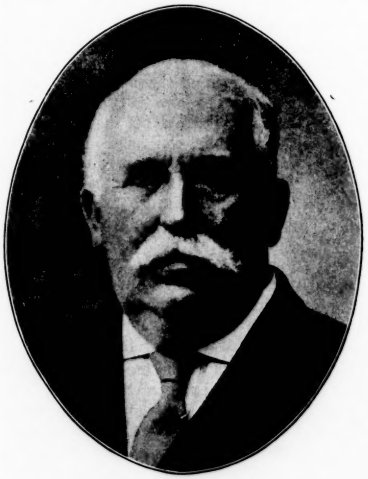
Another portrait is presented, that of David Wil-

liams Cheever, because though officially retired from the active teaching staff he was called on by the students to give lectures on special subjects. He graduated from Harvard College in 1852 and from Harvard Medical School in 1858, where he worked hard in the only laboratory course then given, that of anatomy. His proficiency attracted

DAVID WILLIAMS CHEEVER

the attention of Oliver Wendell Holmes, who appointed him to prepare his prosections and made him demonstrator of anatomy — a position which in those days was the most universally accepted stepping-stone to surgery. Thus he was ripe for the opportunity offered with the opening of the Boston City Hospital in 1864, and was the first surgeon appointed to its staff and performed the first surgical operation. Medical students were attracted to the new institution, and it is a tribute to his success as a teacher that the professorship of surgery should have passed to him from its legendary home, the Massachusetts General Hospital, on the retirement of Henry J. Bigelow. Cheever's teaching was largely clinical at the hospital, but he exemplified the fact that the now somewhat disparaged didactic lecture may be, in good hands, an excellent vehicle of instruction. He was a remarkable lecturer who spoke extemporaneously but after the most careful preparation and digestion of the subject beforehand. He used no oratorical or elocutionary arts. He did not draw on the blackboard and cultivated no mannerisms to awaken attention. He gave a condensed, lucid, simple, epigrammatic word-picture of his subject, so terse and convincing that his hearers could not afford to miss a word. The lecture room was often crowded with men from other exercises or laboratories who gathered as standees behind the seats. The same qualities pervaded his clinical teaching, with the added element of sympathetic understanding which endeared him to both patients and students. He illustrated the doctrine that pedagogy is itself an art, an aptitude for which is born in a teacher and which flourishes with intensive cultivation.

Another surgical teacher who left an indelible impress on his students and other contemporaries was Maurice Howe Richardson, who graduated from Harvard College in 1873 and Harvard Medi-



MAURICE HOWE RICHARDSON

cal School in 1877. He was one of the few teachers of the period, of major reputation, who had neither the alleged disadvantage of the background of a legendary Bostonian nor the advantage of study abroad. He was born in rural Massachusetts in 1851 and approached surgery through the portal of the demonstratorship of anatomy, through which so many subsequently able surgeons had passed. He became a surgeon to outpatients at the Massachusetts General Hospital in 1882, visit-

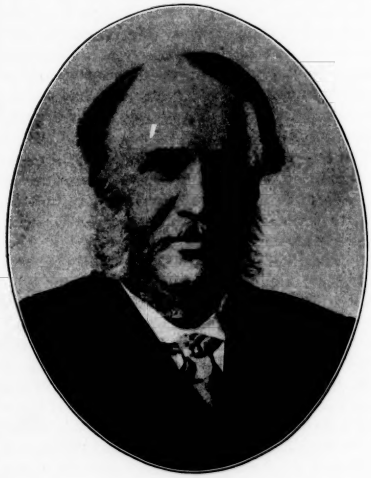
ing surgeon in 1886, assistant professor and professor of clinical surgery and finally Moseley Professor of Surgery, succeeding J. Collins Warren, in 1907. The prophetic glow of the antiseptic era burst into full dawn as he entered the field, and with his native talent, amounting almost to genius, his industry, his knowledge of anatomy, his imagination and his ambition, he reaped its full benefits. One of his first great feats was the removal of a denture, impacted in the lower esophagus, by opening the stomach, dilating the cardiac orifice and extracting the denture with finger and forceps. This was, of course, long before the favoring help of the x-ray. He promptly took up the surgery of the vermiform appendix as a coadjutor of his medical colleague Fitz, and played an important part in aiding the profession to perfect the diagnosis and to establish the necessity for early operation. His talents as a teacher were not conspicuous in systematic lectures but rather in spontaneous clinical exercises and demonstrations. He was endowed with many talents and with the priceless capital of perfect health. He played the piano, the flute, the 'cello and the bassoon; he swam for pastime across Vineyard Sound, and lest that seemed easy because the water was warm, he swam the nine miles from Salem to Magnolia. He made freehand sketches on the blackboard, often simultaneously with both hands. He had an enormous practice, and about him hung the inspiring aura of success. His death at sixty-one was premature but enviable, with no premonitory attacks of pain or incapacity; he retired one evening with a mind full of plans for an early operation on the morrow; when his failure to appear at breakfast caused investigation he was found lying quite peacefully in bed, with the coverlet thrown off by one unconscious motion of the arm, made as life merged suddenly into death.

The dean of the school and the professor of obstetrics was William Lambert Richardson, who came of old New England stock and was born in Boston and educated at Harvard, where he graduated in medicine in 1867, and then studied in Vienna and Dublin. Always a general medical practitioner, he was one of the first to specialize in obstetrics, which he began to teach in 1871 as instructor, finally becoming professor in 1886. Of great executive capacity, he resuscitated the old Boston Lying-in Hospital, housed in a single brick house on McLean Street, enlarged it by the addition of adjoining houses, established its school of nursing and finally saw it move to the present building on Longwood Avenue, in close proximity to the Harvard Medical School, where the private ward bears his name. He made this hospital one of the great institutions of its kind in the world. His was a vigorous personality, of swift accurate sagacity, of few and abrupt words but with the qualities of kindness, cheerfulness, optimism and courage which close acquaintance soon recognized. Richardson, early in his career, lost the sight of one eye through an infection occurring while he was attending a patient with puerperal sepsis; the nurse who assisted him at the same critical case suffered the same calamity as her chief. Students will remember the peculiar fixed expression contributed by the glass eye, and will recall also that so little missed his observation that it was commonly said that "Richardson could see more with one eye than most men with two."

Charles Burnham Porter was born of old New England stock in Rutland, Vermont, in 1840, and having graduated from Harvard College and Harvard Medical School and studied in Europe, approached a surgical career in the classic way through long service as demonstrator of anatomy. He passed through the usual steps in the practice and teaching of surgery at the Massachusetts General Hospital and became professor of clinical surgery. As such he conducted clinical exercises at the hospital and conferences based on theses written by students on particular clinical subjects; he also gave a course on operative surgery on the cadaver at the medical school which, as may be guessed, consisted chiefly of amputations, ligation of vessels, resections of joints and incisions. He was a surgeon of the old precise, anatomical, classical school whose flaps were cut exactly according to rule, and who passed the aneurysm needle always in the direction prescribed by the recognized authorities. In his clinical work, however, he did not allow himself to be handicapped by rules but was progressive and original. His instruction was most useful in inculcating the advantage of meticulous care and accuracy. I once heard him say, in discussing his career, that he had never known what it was to feel tired or ill, and his retirement, when forced by the age limit, seemed unfortunately premature, yet he died suddenly not very long thereafter.

No account of my surgical teachers could be considered complete without mention of John Homans, who, though not a member of the regular surgical hierarchy of the medical school, gave special lectures on pelvic tumors. He was, it may be supposed, one of the Boston legendary type with all its handicaps, being of the third successive generation of doctors. He graduated from Harvard Medical School in 1862 just in time, after a surgical internship at the Massachusetts General Hospital, to enter the Navy as assistant surgeon; later he transferred to the Army and at an

absurdly young age was surgeon-in-chief of the First Division of the Nineteenth Army Corps and medical inspector on Sheridan's staff at the battles of Winchester and Cedar Creek. Then he studied a while in Vienna and Paris and came back to practice surgery wherever he could find it — at the Boston Dispensary, the Children's Hospital, the



JOHN HOMANS

Carney Hospital and finally at the Massachusetts General Hospital. He pitied the fate of women doomed to a life of increasing helplessness and discomfort by the growth of huge ovarian cysts, and encouraged by the success of occasional operators since MacDowell's first operation at Danville, Kentucky, in 1809, and with the safeguards of the new antiseptic method of Lister, he began operating on these patients, first at the Carney Hospital, because the operation was considered by the authorities of the Massachusetts General Hospital to be unjustifiable on account of its danger. Undefeated by early failures and cheered by a rapidly increasing number of successes, he had performed between 1872 and 1900 the prodigious number of over six hundred ovariotomies, as they were called. It will not surprise readers to learn that he had a personality and methods all his own. I remember the final exercises in the course on ovarian tumors which, though occurring in the teaching amphitheater of the medical school, most unexpectedly appeared to be an operation on a living patient. She was wheeled in on a table, fully draped, accompanied by Homans and the assistants in their full surgical costume. It was quite unexpected to see Tom Bonney, technician in the Department of Anatomy, in the role of anesthetist. The incision was apparently made through the skin, subcutaneous tissue, fascia, rectus muscles and peritoneum. Hemostatic forceps were applied and retractors introduced, disclosing a large cystic tumor which was tapped with a Spencer Welles trocar, its fluid contents evacuated, its flaccid sac drawn out of the abdomen and the pedicle ligated and cut away. Then the mounting suspicions of the students were justified by the removal of the sheets and towels, disclosing the patient as a skeleton, in the pelvis of which had been placed a distended pig's bladder and whose abdominal wall had been constructed of suitable layers of fabrics, including red flannel and chamois skin. It sounds like a crude performance and doubtless would scarcely pass muster nowadays, but it was an astonishingly accurate representation of the successive steps of a classic ovariotomy.

Pediatrics was taught our class in the third year by Thomas Morgan Rotch, who justified the characterization as father of modern scientific infant feeding. He was born in New Bedford, but in his veins ran the blood of distinguished medical forebears in Philadelphia. After graduating from Harvard College and from Harvard Medical School in 1874, he served as medical house officer at the Massachusetts General Hospital, studied for two years in Berlin, Vienna and Heidelberg, and on his return joined, in turn, the staffs of the Boston Dispensary, the Boston City Hospital, the Children's Hospital and the Infants' Hospital. Lectures on "Certain Diseases of Children" had been given by various incumbents of the chairs of medicine before this, but Rotch's lectures on "The Prognosis, Diagnosis and Treatment of Diseases in Children" emphasized the specialized nature of the subject and led to the establishment, at Harvard Medical School, of the first professorship in pediatrics in this country, to which Rotch was appointed first as assistant professor and later, in 1893, as full professor. It was the pioneer department in America — well organized and conducted — and led to the establishment, in 1891, of the first laboratory in the country for the modification of milk for babies. Rotch published his standard textbook on pediatrics in 1895, and wrote monographs on pericarditis and the roentgenology of bones during growth. His campaign for clean milk and other methods to promote safe artificial infant feeding were at first derided and opposed, but he lived to see them adopted. Invalidism in his immediate family and the tragic loss of an only son might have ruined the career of a less

brave man, whose rather gentle and exquisite personality perhaps failed to give the impression of the courage and determination which helped him reach his objectives.

A useful man, an important factor in the effective organization of surgical teaching, was Herbert Leslie Burrell, professor of clinical surgery at the Boston City Hospital; without some of the advantages of his colleagues both in pre-medical education and European study, he succeeded by hard work and ability in bringing to the surgical department some attributes which it might otherwise have lacked. He was a born organizer and demonstrator, and supplemented the work of his more showy fellows to the end that the students might have a comprehensive and complete course. He was a close student of the science of surgery, but was somewhat lacking in the instinctive talents which are vouchsafed to some. He performed successfully such unusual operations as ligature of the innominate artery by meticulous preparation and anticipation of every difficulty. He was prominent in many branches of public service, and in the Spanish-American War he transformed an old freighter into a model hospital ship, the *Bay State*, which was inspected and adopted as an example by foreign observers, and in command of which he made three trips to Cuba. The students smiled at his sententiousness and formality and respected his devotion to the work of teaching them. His untimely death revealed that the handicaps of undeclared illness might have thwarted a less determined man.

It is hard to realize that such an important and sharply defined field as neurology was first mapped out by one of the teachers of this era — James Jackson Putnam. He, too, was a legendary Bostonian, a grandson of James Jackson, cofounder of the Massachusetts General Hospital and first professor of clinical medicine. Educated at Harvard and a house officer in 1869 at the Massachusetts General Hospital, he studied in Leipzig and Vienna under Rokitansky and Meynert and in England under Hughlings Jackson. On his return he began to lecture in 1872 on nervous diseases and established the first neurological clinic at the Massachusetts General Hospital in one small room of the Out Patient Department. He became a leader in this untried field, and for him was created the chair of diseases of the nervous system. His facilities were so meager that he conducted a neuropathological laboratory in his own home, where he made pioneer studies in organic neurology. As a teacher in a rather difficult field for junior students he was not ideal, since his mind ranged so widely over the various related aspects that it was difficult for him to be a clear expositor of fundamental principles. As he went on he became much interested in the functional neuroses and psychoanalysis and in speculative philosophy, which he pursued with James, Royce and Bergson. His Shattuck Lecture of 1899, entitled "Not the Disease Only, but Also the Man," is a masterpiece of the doctrine suggested by the title. Like most of his race he was a useful citizen — always ready to serve in the charitable and public enterprises of his fellow-citizens. He had the mental qualities of a man of science and a philosopher. His students appreciated the importance of his teaching more in retrospect after they had personally met the problems of neurology in their experience as practitioners.

It is impossible in the brief time at our disposal to acknowledge in full my debt in the matter of my education, but at least brief tribute must be paid to teachers whose stature was no less than those already mentioned, but whose teaching covered lesser fields. James Clarke White, born in Maine in 1833 of vigorous Ulster stock, and educated at Harvard, was a man of versatile intellectuality embracing natural history, literature and art. After graduation in medicine and as house pupil at the Massachusetts General Hospital in 1856, he studied in Vienna, realizing that Paris had passed its heyday as a medical center. On his return he taught chemistry at the school, joined the medical staff of the hospital, noted the neglect of diseases of the skin and cultivated that study with characteristic genius. For him was created the first chair of dermatology in this country, in 1871, which he held for over forty years. He continued his interest in natural history and prepared an herbarium of the wild flowers of New England; he edited the *Boston Medical and Surgical Journal;* he traveled; he cultivated esthetic interests and literature. His constant influence was toward elevating medical education in general and dermatology in particular, and he was one of the group of younger men on whom President Eliot depended for support in his educational reforms. I remember him as tall, erect, vigorous and white-bearded, conducting his skin clinic with military discipline and efficiency. He raised a neglected branch to the dignity of an important special field.

Oliver Fairfield Wadsworth taught me ophthalmology. He was a legendary Bostonian, born in 1838, educated at Harvard and the Massachusetts General Hospital, and saw service as assistant surgeon, Fifth Massachusetts Cavalry, during the Civil War. At its close he studied ophthalmology for two years at Zurich, and on his return became "visiting ophthalmologist to all the hospitals in Boston where there was an eye service." He was a conscientious teacher and a useful citizen, espe-

cially in the medical community. He is said to have been the first to introduce clinical as well as written examinations into the school; he helped organize the Boston Medical Library and was its clerk and secretary for thirty-six years. He was an affectionate, disputatious, cheerful man whose modest personality gave all too little opportunity to the students, during a too brief relation, to appreciate his worth.

Charles Montraville Green was William L. Richardson's right-hand man in the Department of Obstetrics, and long after my school days succeeded to the professorship of obstetrics, with the added field of gynecology. At the turn of the century he was supplementing his chief's lectures by his characteristically well-planned conferences and practical exercises. His summer course in obstetrics, first established in 1880, was famous for its effective organization and success in affording students an experience at least remotely comparable to a service as intern in a lying-in hospital. Green was a lucid, conscientious, painstaking teacher and able clinician, whose service to the medical school for thirty odd years and on the staff of the Boston City Hospital for forty-four years influenced the education of an army of living pupils.

Edward Hickling Bradford taught orthopedic surgery. Born in Boston in 1848, he was a vigorous scion of Pilgrim stock, a direct descendant of Governor William Bradford. Educated at Harvard College, Harvard Medical School and the Massachusetts General Hospital, he studied abroad for two years, and returning joined the surgical staffs of the Boston City Hospital and the Children's Hospital, where the new specialty of orthopedic surgery attracted his interest. Instruction in this branch was confided to him in 1881, and subsequently he became the first professor of orthopedic surgery. He was a man of gentle but forceful personality, characterized by integrity, devotion to his work, fairness and constancy in all his relations. In his early prime a bicycle accident caused the loss of an eye and serious disfigurement, which with characteristic absence of self-pity he refused to permit to handicap him. For six years, beginning when sixty-four years of age, he acted as dean of the school, during the trying period which included the World War. When at seventy-five his remaining eye gave out, he learned Braille and continued his quiet, unassuming, indomitable service to the community. Another great example of *noblesse oblige* was Bradford.

Clarence John Blake, professor of otology, was one of the first in this country to make a specialty of otology after the invention of the forehead mirror, and studied under the great Politzer. He was a kind, responsive and ingenious man whose teaching was replete with interest and fascination far beyond the capacity for appreciation of students in an elementary course.

Franz Pfaff, exotic, bearded, bespectacled Teuton, taught pharmacology with emphasis on the experimental aspect. Frank Winthrop Draper, scholarly, modest, hard-working, dependable, a veteran of the Civil War, was appointed the first medical examiner of Suffolk County at the time of the abolishment of the old coroner system and taught legal medicine, the while he served the State on the Board of Health, the Boston City and Children's hospitals as visiting physician, and organized medicine as secretary, treasurer, orator and president of the Massachusetts Medical Society. Davenport in gynecology, Harrington in hygiene, Hills in chemistry and others too numerous to mention (and omitting the names of those still living) played their lesser but important parts in this educational complex.

A comparison of the medical education at the turn of the century with that of forty years later, when I have completed my allotted contribution to the same cause, would be too long a study, but certain outstanding characteristics are perhaps worth emphasizing. The teaching staff was largely recruited from Boston and New England, and indeed from Harvard University itself. None but the pre-clinical laboratory branches were in charge of full-time teachers, whose salaries were modest enough. Most of the teaching staff of professional rank belonged to that group of legendary Bostonians who had in varying degrees the "crushing disadvantages of an assured position, binding traditions and a competence" before referred to; yet it is my opinion that their teaching bears comparison both in substance and in spirit with that of their successors of today. Forced — or perhaps privileged — as they were to gain their livelihood from private practice, they nevertheless felt a pride and an interest in their academic responsibilities which made it unthinkable that they should slight them. The young physicians whom they educated to go out into the community to heal the sick learned the art from men who were spending their lives in the same pursuit. Fortunate was the school of the turn of the century that its habitat was a community where, in spite of its comparatively slender resources, it could draw its intellectual and spiritual sustenance from citizens whose ideals and standards of education and public duty were those of colonial New England.

Free use of existing biographical material has been made and is hereby acknowledged.

193 Marlboro Street.

INTRA-ABDOMINAL TORSION OF APPENDICES EPIPLOICAE

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BOSTON

TORSION of an epiploic appendix may give rise to acute abdominal symptoms which result in emergency operations. The diagnosis is difficult and is rarely made. The first 16 cases reported in the literature were abstracted by Hunt[1] in 1919. He gave a general discussion of the subject, and included abstracts of cases of appendices epiploicae acting as foreign bodies and also their relation to hernias.

In 1936 Fiske[2] reviewed the literature and found 26 additional cases, making a total of 42. He added 2 cases from his clinic, in both of which a correct preoperative diagnosis was recorded. Since his article appeared 2 other cases have been reported,[3] making a total to date of 46 recorded cases.

Appendices epiploicae are small pouches of peritoneum filled with fat, and are found in one or two rows along the large intestine. On the ascending and descending portions they are located medially, while on the transverse colon they are on the lower part. They are pedunculated and are supplied with small arteries and veins. Their function is not known. They act as a storage place for fat, and they may also facilitate the motion of the large bowel by acting as skids or cushions.

Disease of these structures is very limited. The commonest lesion is twisting of the pedicle with resulting infarction. Infarction, gangrene and strangulation are terms encountered in this connection. The necrotic tab may, perhaps, by becoming attached to the bowel wall give rise to severe, cramp-like pain.

The onset of symptoms in cases of infarction is usually sudden and without an obvious predisposing cause. Most frequently the pain is generalized but may become localized. It is sometimes associated with nausea and vomiting. The portions of large bowel involved are about equally divided between sigmoid and cecum.

On examination the patient does not appear ill. He may show signs of discomfort from steady aching or peristaltic pain. Rectal examination may show tenderness on one or both sides. The temperature is only slightly elevated, if at all, but the white-blood-cell count may reach 20,000.

Diagnosis is difficult. The entire picture when the pain is on the right side is impossible to distinguish from that of a mild attack of appendicitis. Among the diagnoses made preoperatively have been cholecystitis, ovarian cyst with twisted pedicle, renal calculus, diverticulitis and intestinal obstruction. When the lesion is on the left side it is more easily recognizable.

Treatment is removal of the necrotic mass after ligating its base, and careful inspection of the bowel wall in order to see that no perforation exists. In one case the mass was left in because of inaccessibility. The patient continued to have pain, spasm and tenderness for ten days.

I have recently seen a patient (Case 6) on whom a preoperative diagnosis of subsiding appendicitis was made but at whose operation a necrotic twisted epiploic appendix was found. In order to determine the frequency of occurrence of this condition I have examined the case records of the Massachusetts General Hospital and have found only 5 other cases. In none of these was a correct diagnosis made.

CASE 1 (W. S. 175356). The patient, a 36-year-old man, was admitted April 4, 1911. Twenty-four hours previously he had developed sudden severe pain in the right lower quadrant of the abdomen. There was no radiation and no nausea or vomiting. The patient went to bed and applied an ice bag. He had chills and fever all day.

Examination on admission showed slight spasm in the right upper quadrant. There was marked spasm in the right lower quadrant, with some tenderness. No masses were felt. The temperature was 99°F., the pulse 62, the respirations 20, and the white-cell count 12,600. The urine was normal.

The preoperative diagnosis was appendicitis. At operation a necrotic mass the size of a small apple was found on the cecum. This was removed and an appendectomy was performed. The pathological laboratory reported peri-appendicitis and infarction of an epiploic appendix.

CASE 2 (E. S. 193799). The patient, a 28-year-old woman, was admitted February 19, 1914. She had had an attack of pain in the bladder region 16 years previously, of 5 days' duration. Four months later, she had fever, accompanied by pain, in the left upper quadrant of the abdomen, for 2 weeks. There was some nausea and vomiting. Several subsequent attacks of pain in the abdomen occurred, coming at night and lasting for 1 hour. Three days before admission the patient was awakened by pain in the right lower quadrant. There was no radiation, but the pain was severe enough to double her up. It gradually diminished but fluctuated in intensity. There was no nausea or vomiting.

Examination revealed a round, soft, tympanitic abdomen. There was marked tenderness in the left flank and slight tenderness in the right lower quadrant. No masses and no spasm were felt. An appendectomy was per-

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formed and a calcareous epiploic appendix was removed from the sigmoid. The pathological laboratory reported chronic appendicitis and gangrene of an epiploic appendix.

Case 3 (E. S. 196336). The patient, a 32-year-old man, was admitted June 30, 1914. About 28 hours previously he had begun to have sharp pain throughout the lower abdomen. This lasted about 2 hours and was accompanied by nausea and vomiting. He went to work the following day but had another attack in the afternoon, lasting 1 hour. A few hours later he had a third attack, which was more severe than the previous two. It was characterized by severe knife-like pain in the right lower quadrant of the abdomen.

Examination revealed a soft, tympanitic abdomen. There was no spasm or rigidity. There was tenderness in the left lower quadrant. There was also some pain over McBurney's point. No masses were felt. Rectal examination showed tenderness on both sides. The temperature was 99.2°F., the pulse 100, the respirations 20, and the white-cell count 20,000. The preoperative diagnosis was acute appendicitis.

Exploration revealed a slight kink in the vermiform appendix but no inflammation, and an inflamed infiltrated mass attached to the sigmoid. Because of its inaccessibility it was not removed. An appendectomy was performed. The postoperative diagnosis was strangulated epiploic appendix. The patient continued to have pain with some spasm in the left lower quadrant for 10 days.

Case 4 (W. S. 305485). The patient, a 52-year-old woman, was admitted January 18, 1935, complaining of soreness in the right lower quadrant of the abdomen. This had come on suddenly about 24 hours before entry. She took a laxative and had two small bowel movements, without any increase in pain. There was no nausea, and no urinary symptoms. The pain persisted but was not severe enough to keep her awake or interfere with her appetite.

On examination the abdomen was pendulous and there was a tender point to the right of and below the umbilicus. There was some tenseness of the muscles. The rectal and pelvic examinations were negative. The temperature was 98.6°F., the pulse 86, and the white-cell count 10,200. The urine was normal. The preoperative diagnosis was acute appendicitis.

At operation a strangulated epiploic appendix was found over the base of the vermiform appendix. Both were removed. The postoperative diagnosis was infarction of an epiploic appendix.

Case 5 (E. S. 14999). The patient, a 46-year-old man, was admitted January 27, 1937, complaining of generalized abdominal pain of 30 hours' duration. There was no nausea or vomiting, but the patient could not eat because of persistent abdominal cramps. He took a tablespoonful of castor oil and was awakened in the night with generalized cramps. A second dose was followed in 2 hours by a profuse bowel movement, the first in 2 days. The cramps persisted and the patient was admitted to the hospital.

On examination the abdomen was soft. There was some distention of the cecum and ascending colon. There was no mass or spasm. There was localized tenderness in the right lower quadrant. Rectal examination showed tenderness, high on both sides. The temperature was 99.5°F., and the white-cell count 15,200. The urine was normal. The preoperative diagnosis was possible partial obstruction, possible diverticulitis and possible acute appendicitis.

Exploration showed the vermiform appendix to be normal. On freeing the cecum a loop of ileum was brought up, and a piece of hemorrhagic tissue measuring 3 by 4 cm. was found attached to it. It had apparently become separated from the colon and attached itself to the ileum. It was readily freed, and the wound was closed without drainage after removal of the appendix. The pathological laboratory reported infarction of an epiploic appendix and periappendiceal hemorrhage.

Case 6 (private case). The patient, a 20-year-old man, was seen January 17, 1938. He had been perfectly well until 30 hours previously, when after eating a small, hur-

Table 1. *Summary of Clinical Data.*

Case No.	Age	Sex	Pain location	Pain duration	Other Symptoms	Temperature °F.	White-Cell Count $\times 10^3$	Local Signs	Preoperative Diagnosis	Location of Lesion	Treatment	Results
1	36	M	R. L. Q.	24 hr.	Chills	99	12.6	Spasm	Appendicitis	Cecum	Appendectomy and removal of epiploic appendix	Relieved
2	28	F	R. L. Q.	16 yr.	Nausea, vomiting	99.4	—	Tenderness	Appendicitis	Sigmoid	Appendectomy and removal of epiploic appendix	Relieved
3	32	M	Generalized	28 hr.	Nausea, vomiting	99.2	20.0	Tenderness (L. L. Q., R. L. Q.)	Appendicitis	Sigmoid	Laparotomy	Pain and tenderness, with spasm, in left lower quadrant, for 10 days.
4	52	F	R. L. Q.	24 hr.	None	98.6	10.2	Tenderness (R. L. Q.)	Appendicitis	Cecum	Appendectomy and removal of epiploic appendix	Relieved
5	46	M	General	30 hr.	None	99.5	15.2	Tenderness (R. L. Q.)	? Intestinal obstruction, ? diverticulitis, ? appendicitis	Attached to ileum (origin doubtful)	Removal of necrotic mass adherent to ileum	Relieved
6	20	M	L. L. Q.	30 hr.	None	99.2	11.2	Tenderness, spasm (L. L. Q.)	? Appendicitis	Sigmoid	Appendectomy and removal of epiploic appendix	Relieved

ried lunch he immediately felt a dull pain in the left lower quadrant of the abdomen. This persisted through the night and gradually became worse. The pain was not severe enough to double him up but was enough to make him consult his physician. There was no nausea, vomiting, urinary disturbance, change in bowel habits or melena. The patient had had no previous similar attacks. The past history was negative except for removal of a semilunar cartilage 1 year previously.

Examination revealed a well-developed and healthy-appearing man. The abdomen was flat. There was slight tenderness over McBurney's point. In the left lower quadrant there was marked tenderness with muscular spasm. There was also slight spasm in the right lower quadrant. There was no pain on leg-raising, but some pain on the left with hyperextension of the right leg. Rectal examination showed some tenderness on both sides, but it was more marked on the left. There was some fecal material in the rectum. The urine was normal. The temperature was 99.2°F., the pulse 84, the respirations 20, and the white-cell count 11,000.

It was thought that appendicitis was likely, although the symptoms were somewhat atypical, and the patient was admitted. An enema caused some pain and was discontinued. The white-cell count a few hours later was 9600. The following day the enema was repeated with less discomfort. The white-cell count dropped to 6300, and the pain and tenderness were much less marked. A flat x-ray film of the abdomen was negative. In the afternoon the pain and tenderness increased and the pain became peristaltic in character. A barium enema was given. There was some pain when the colon was partially filled, but spasm and diverticula were not demonstrated. The appendix was not seen. The patient was then given a motor meal and x-ray photographs were taken at intervals. No Meckel's diverticulum was seen. The appendix was visualized and was seen to contain many fecoliths. In view of the fact that the patient was employed on a ship, it was decided to explore the abdomen.

On opening the peritoneum a firm nodule measuring about 2 by 2 cm. was felt in close relation to the sigmoid. It was delivered without difficulty and found to be a gangrenous epiploic appendix. It had become twisted on its pedicle and was adherent to the sigmoid. This mass was freed and its pedicle ligated and excised. There were some adhesions around the cecum. A kinked and thickened vermiform appendix was removed.

Convalescence was uneventful, and there has been no reoccurrence of symptoms.

Presentation of 6 additional cases of torsion of appendices epiploicae brings the total reported to 52. There were 29 men and 23 women. The age incidence varied from twenty to seventy; the average age of this series is thirty-five. In the published cases there were 5 in whom the condition was an incidental finding. In the others, acute symptoms were present which necessitated operation.

Pain is the commonest symptom. It is usually sudden in onset and varies from moderate to severe. It is often peristaltic in character. Localized tenderness may be present, with or without spasm. The temperature is slightly elevated, and the white-cell count may reach 20,000.

There were 6 deaths in the cases reported in the literature. In 2 of these the torsion was an incidental finding at autopsy, 1 patient having died of acute hemorrhagic pancreatitis and 1 of cardiac failure. In 4 cases, however, death was definitely related to torsion of an epiploic appendix. In 1 case reported by Riedel[4] the patient had intestinal obstruction and died from peritonitis the day after operation. In Ebner's[5] case death occurred on the seventh postoperative day and was due to paralytic ileus secondary to interference with the blood supply from the torsion. Seelye[6] reports a death due to generalized peritonitis a few days after drainage of an abscess caused by a twisted gangrenous epiploic appendix. Baumeister, Hargens and Morsman[7] report a death from intestinal obstruction due to adhesions between appendices epiploicae.

SUMMARY AND CONCLUSION

Only 46 cases of torsion of an epiploic appendix have been reported.

The symptoms and signs of appendicitis are closely simulated.

The preoperative diagnosis is rarely made.

Six additional cases are summarized.

Vague abdominal pain is probably often due to an unrecognized strangulated epiploic appendix.

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LUNG ABSCESS*

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BOSTON

ANY disease which is chiefly a complication of other medical and surgical conditions, and therefore presumably in some measure controllable by the management of those conditions, deserves exhaustive investigation, especially when the disease still gives rise to a reported mortality in treated cases of 30 to 50 per cent.[1, 9, 22, 37]

Diagnosis of nontuberculous abscess of the lung is relatively simple. It is rather a problem of treatment and, even more significantly, of prevention. Intelligent treatment is based on an accurate understanding of the pathology and the pathologic physiology involved, and any program of prophylaxis must presume some knowledge of etiology, bacteriology and precipitating incidents.

ETIOLOGY

There are three possible pathways of infection to the lung parenchyma besides direct contamination incident to trauma. These are the trachea and bronchial tree, the blood stream and the lymphatics. It seems fairly certain that the lymphatic route must be rare, though occasionally it may be of significance in bringing infection from a focus in surrounding tissue. That infection frequently reaches the parenchyma through the bronchi is generally conceded, and it seems probable that it is carried there by the blood stream more often than is clinically suspected.

Cutler and his associates[10, 11] found many years ago that they could produce lung abscess in dogs by releasing in the venous circulation an embolus consisting of a small section of a tiny vein filled with organisms. Their findings led them to believe that embolism from the operative wound and local immune reactions in the lung were the important factors in postoperative lung abscess. Fetterolf and Fox[14] studied tonsillectomy wounds in dogs and found hemorrhages, thrombi, necroses and bacteria present in the surrounding tissue. Because the muscular pharynx can never be put at rest, they believed that thrombi were readily discharged, resulting in pulmonary infarcts which subsequently became infected with organisms from the upper respiratory tract. Many of these pulmonary infarcts probably escape notice because of their small size, sterile character and prompt resolution. Castleman[6] and Hampton[19] on the basis of correlated postmortem x-ray and anatomical studies, have expressed the belief that incomplete pulmonary infarction is much more frequent than was previously suspected.

Recent investigators have been able to produce lung abscess readily by injecting infected material into the bronchi. Weiss,[38] for instance, has done it repeatedly in monkeys by inserting a mixture of agar and anaerobic organisms obtained from lung abscesses at operation into the bronchus of the left lower lobe. His evidence suggests that aspiration of infected material, together with plugging of the bronchus, is responsible for abscess formation.

Clinically it is often impossible to establish whether an embolus or aspiration is the origin, but it seems probable that the latter route is usually responsible. The bacteriological flora of a well-developed abscess is qualitatively very similar to that of the unhygienic oral cavity. It is difficult to see how a septic embolus, from a laparotomy wound, for instance, could give rise to this picture in itself. Where emboli are involved, a sterile or simple primarily infected infarct probably results, and this is secondarily infected by a complication of buccal organisms.

In analyzing the cases of lung abscess at the Boston City Hospital, Freedman[15] concluded that pulmonary atelectasis was the important factor in stagnation of infected material in shut-off portions of the lung. He found that chronic pulmonary conditions such as bronchitis, asthma and bronchiectasis provided fertile soil for acute suppuration. Apparently there must be a primary lesion of some kind in the lung before secondary infection by mouth organisms can take place. This lesion may be due to infarction as a result of an embolus, or atelectasis due to bronchostenosis, or obstruction by plugs of secretion. The normal bronchial tree is admirably equipped by its secretions, cilia and reflex cough mechanism to handle the organisms which must constantly descend from the upper respiratory tract, but when this process is disrupted by mechanical factors incident to disease or surgical manipulation, infection is liable to ensue.

*Boylston Prize Essay for 1939; read before the Boylston Medical Society, Harvard Medical School, February 20, 1939.

PATHOGENESIS

The numerous series of cases of lung abscess reported in the literature all show that the majority of cases follow operations on the upper respiratory tract and pneumonia. Of the 276 cases reported by Freedman,[15] 196 were sequelae of medical conditions and 76 followed operative procedures. The medical causes included pneumonia, chronic cough, influenza, tuberculosis and carcinoma. A large percentage of these cases could ultimately be traced back to the common cold or some other acute process with an intervening atelectasis in the lung. Great numbers of these patients were in poor general condition owing to alcoholism, diabetes or cardiac or peripheral vascular disease, and poor oral hygiene was practically universal. The incidence of lung abscess as a postoperative complication of various operations in this series is shown by the following ratios:

TYPE OF OPERATION	RATIO	
Tonsillectomy and adenoidectomy	1:1654	(1:626 in adults)
Tooth extraction	1:320	
Appendectomy	1:1411	
Pelvic surgery	1:774	
Gastric surgery	1:54	
Gall-bladder surgery	1:258	

The high incidence of this condition following operations on the upper abdomen, namely the stomach and gall bladder, is especially interesting because it is also true of atelectasis, massive collapse, embolism and other postoperative pulmonary complications.[21]

Of the cases of lung abscess at the Massachusetts General Hospital reported by King and Lord,[22] 56 per cent followed operations on the upper respiratory tract and 9 per cent other operations; 23 per cent were insidious in onset, and 8 per cent had a stormy onset following pneumonia. In a series reported from Toronto,[37] 30 per cent followed upper respiratory operations, 19 per cent followed pneumonia, 7 per cent were associated with definite pulmonary emboli and 33 per cent were classed as idiopathic. These percentages are representative of many reported series.

The etiologic factors in cases in children have a somewhat different proportion. On examining records of the cases diagnosed as lung abscess at the Boston Children's Hospital during the ten years previous to 1938, it was found that 53 per cent followed tonsillectomy but none followed any other operations, 6 per cent followed the aspiration of foreign bodies and all the rest developed from unresolved pneumonia or severe upper respiratory infection. In all these cases in children it was possible to find some definite precipitating incident. This is a very interesting fact. The bacteriological flora of the buccal cavities of children under ten differs from that of adults in that fusiform bacilli and spirochetes are rarely found and staphylococci are more prevalent.[24] In children, oral hygiene is much better and the severe gingivodental infections of the adult ward patient are rare.

Stern,[36] at the Mount Sinai Hospital in New York, examined all the cases of lung abscess in which the etiology was unknown, and found that 84 per cent presented a typical and uniform picture of marked dental disease with infected gums and large masses of dental tartar. It is in dental tartar that anaerobic organisms thrive. The tartar when aspirated, Stern believes, acts as an irritating foreign body and therefore facilitates the development of lung suppuration in the presence of infecting organisms. His observations seem sufficiently well controlled to permit the conclusion that oral disease is disproportionately common in patients with abscess of the lung of unknown etiology. Neuhof[30] found gross gingivodental infection in 17 of 26 cases of lung abscess of unknown etiology, and pointed out that among the children in his series where this type of infection was not present there were no cases of unknown etiology. There are other reports[39] with the same clinical impression, so that it seems probable that many cases of so-called idiopathic abscess are due to inhalation of infected material from badly diseased teeth and gums.

In many cases it is difficult, if not impossible, to determine whether a sudden illness is due to an abscess proper or to a pneumonic condition with subsequent abscess formation. The roentgenologist may not be sure about the consolidation he first sees, and physical signs, if present at all, are not diagnostic. It is the patchy bronchopneumonia of the influenza type that seems particularly prone to give rise to lung abscess.

Lung abscesses due to the aspiration of foreign bodies usually occur in children, and need no comment here, except to call attention to the interesting observation in a British journal[32] that the probable reason why these cases are so much commoner in the United States than in England is the popularity in this country of chewing gum and peanuts.

PATHOLOGY

Single lung abscesses are generally located in the right lung. This is also true, however, of other pulmonary lesions such as embolism and lobar pneumonia. The more direct continuity of the right primary bronchus with the trachea is of interest in this regard. Also the right branch of the pulmonary artery is slightly larger and is said to

come off more directly from the bifurcation, and the right lung itself, of course, is larger than the left.

Most abscesses are situated at the periphery of the lung, and the overlying pleura very early shows clouding and thickening. Neuhof[30] believes that even in the early acute stage pleural adhesions are present. He describes the lesion as at first an intense necrotizing inflammation of the affected bronchioles with rapid destruction of lung tissue, resulting in cavitation with contained pus and detritus. Others believe that there is first an area of diffuse pneumonitis, which either resolves of itself or undergoes necrosis, liquefaction and cavity formation.[12, 23, 24] Either of these processes is in contrast to that stimulated by the tubercle bacillus, which is essentially fibrosis. If the abscess is recent and progressing, its wall consists of soft, necrotic lung tissue surrounded by edema and hyperemia. The dense rind of the abscess by x-ray, frequently described as fibrosis, melts away so completely under satisfactory treatment that it must be exudative.[33]

Single abscesses are usually round and contain thick, yellowish or greenish pus, which may or may not be blood-streaked. It may be odorless, sweet or foul, depending on the particular organisms present. If the cavity can be successfully drained, it may be obliterated except for a thin, fibrous scar. On the other hand, if chronic pulmonary suppuration and gangrene ensue, the sputum is apt to become thin, gray or brownish green and intensely foul, and in that event usually reveals fusiform bacilli and spirochetes in large numbers. There is progressive ulceration of lung tissue, so that the characteristic lesion is ragged, with an advancing periphery and enlarging cavities filled with necrotic lung, exudate and innumerable bacteria. Patients with these lesions die from absorption of bacterial toxins if they survive any attempted operative procedures and escape such complications as massive hemorrhage, metastatic brain abscess and meningitis. They may drown in their own pus owing to the rapid bronchiogenic spread of the disease.

BACTERIOLOGY

In lung abscess, as in spreading peritonitis following perforation of the bowel, there is usually more than one organism present and the flora may be very complex. The bacteria found in any case depend on whether material is smeared or cultured from the sputum, from pus obtained bronchoscopically or from pus obtained directly from the abscess cavity at operation. Bucher[5] cultured pus obtained bronchoscopically from 118 cases and found the following organisms predominant:

ORGANISM	NO. OF CASES
Streptococci (all varieties)	93
Micrococcus catarrhalis	61
Pneumococci	50
Haemophilus influenzae	41
Staphylococcus albus	39
Staphylococcus aureus	22
Diphtheroids	29
Spirochetes	25
Fusiform bacilli	25
Micrococcus tetragenus	22

He cultured 20 of these anaerobically and found 14 positive for streptococci. Cohen[7] found anaerobic streptococci in all 16 cases cultured at operation, and in 14 of these he found *Bacillus melanogenicum*. The latter organism has also been widely cultured recently from cases of peritonitis and has been thought[2] to be the agent responsible for the fetid odor. Neuhof and Wessler[31] believe that all putrid abscesses are initiated and certainly maintained by pathogenic anaerobes. They state that they can be found at every stage of the infection and disappear when the infection subsides. Supporting the pathogenicity of these organisms is the work of Cohen,[8] who found that washed filtrates of 80 per cent of anaerobic gram-negative bacilli obtained from lung abscesses at operation gave a positive Shwartzman reaction. Only with organisms giving this reaction was he able to produce severe necrotic lesions in the lungs of rabbits.

There really is no need to postulate a single bacteriologic etiology. We know that many types of organisms give rise to pneumonia, for instance. Possibly the original infecting agent has no clinical significance other than producing the minor lesion in the lung which paves the way for secondary invaders which are in turn responsible for the characters of the full-blown abscess. Possibly it is the quantitative relation between these various secondary organisms that is responsible for differences seen in the clinical picture and course of the disease in various patients. The importance of the synergistic action of various combinations of bacteria we know very little about. From the work of Meleney, Olpp, Harvey and Jern[27] and others on the bacteriology of peritonitis, it seems probable that synergism often enhances remarkably the pathogenicity of given organisms in the body. The importance of the various types of fungi frequently found is also little understood.

DIAGNOSIS

The clinical picture is so well known that it is necessary here only to recall the typical onset, one to three weeks following operation or pneumonia, of cough, at first dry, and then productive of sputum, which may or may not be foul and may or may not be blood-streaked. There may be dysp-

nea, cyanosis or chest pain of a pleural nature, or these may all be absent. A rise in temperature with general malaise and weakness is a fairly constant finding. Physical examination is seldom of much aid early in the course of the disease. Localized signs in the chest vary according to the size and content of the cavity and the amount of surrounding exudation. Fever, leukocytosis and other constitutional signs of sepsis are present. There may be clubbing of the fingers.

The picture at any one point in the course of the disease depends primarily on the adequacy of drainage, whether by the bronchus or through a surgically created tract. If drainage becomes inadequate the temperature and white-cell count rise, weakness becomes more marked and the patient appears to be losing ground until drainage is reestablished.

X-ray examination may show a characteristic single localized abscess cavity with a fluid level, surrounded by a narrow margin of pulmonary infiltration, or the cavity may be filled so that its shadow merges with that of the surrounding exudation in the lung. X-ray examination in the upright position is essential to the demonstration of fluid levels. The abscess may be masked by the simultaneous presence of empyema. While x-ray examination may be important for primary diagnosis, it is of even greater significance in accurate localization of the abscess, which is so necessary for satisfactory postural or surgical drainage. Stereoscopic films and films taken in three planes are invaluable to this end. Sometimes visualization of the bronchial tree by Lipiodol gives the most accurate picture, although it is notably difficult to introduce Lipiodol into an abscess cavity by way of the bronchi. X-ray examination is also of great aid in following the course of the disease, estimating the adequacy of drainage and helping to decide when surgery should be instituted.

Another aid to diagnostic localization is bronchoscopy. When it is skillfully performed, the exact bronchopulmonary segment from which pus is coming can be located in almost every case. Bronchoscopy is all-important where a foreign body or tumor is suspected, and as an aid in evaluating the cause of impaired bronchial drainage.[34]

TREATMENT

No program of therapy can be outlined which is more than generally applicable, and at present it seems probable that the ultimate result depends less on the choice of method of treatment than on the thoroughness and intelligence with which the method selected is carried out. However, some systematic plan, labile enough to fit the demands and complications of the particular case, should be adopted. The internist and the surgeon must work together from the beginning, because the rational treatment of lung abscess involves the methods and judgment of both. Every case should have a trial of medical therapy before surgery is instituted, but the surgeon should follow the case with the internist during this period.

Of 2114 cases collected from the literature by Allen and Blackman,[1] 51 per cent were treated conservatively and 49 per cent came to some type of surgical procedure. The mortalities in these two groups are almost identical; 34.4 and 34.2 per cent. The difference in the type of the cases in the groups should be remembered. It is doubtful whether any lung abscess heals without the establishment of good drainage. This may be accomplished, however, by spontaneous rupture into a large bronchus as well as by various medical and surgical procedures. This appears to be particularly true in children. One third of the cases diagnosed as lung abscess on the basis of cavitation by x-ray at the Children's Hospital cleared with only conservative therapy in two or three months, and most of these were symptom-free after six weeks' observation. This high percentage suggests that there are many more cases of mild lung abscess which clear up spontaneously at home and never get diagnosed. Graham, Singer and Ballon[18] hold the same view as to adults, believing that almost every patient with streptococcal pneumonia has some small abscesses. In adult series probably 1 case in 5 heals spontaneously. Expectation for this type of recovery is largely confined to patients with a short previous duration of the disease. The exciting cause of the abscess appears to be of no significance.

What is the proper medical treatment of lung abscess? Besides complete bed rest, a high-vitamin, high-caloric diet, adequate fluids and pleasant surroundings, the importance of all of which cannot be overestimated, medical measures include postural drainage, bronchoscopic drainage, pneumothorax, the giving of arsenic and guaiacol and symptomatic drug therapy. The regimen should be as strict as that of pulmonary tuberculosis, and cases which respond should receive hospital or sanatorium care for considerable periods after they have become symptom-free. This step is only too often neglected, with a discouraging recurrence of the lesion.

Postural drainage is the most important single procedure. In order to be of value, it must be carefully and faithfully carried out. The exact location of the abscess must be determined, so that the best position for drainage can be assured. "Posturing" is usually advocated for fifteen or twenty minutes three or four times a day. It is

not infrequent to obtain 300 or 400 cc. of pus per day by this method. Continuous postural drainage is too uncomfortable. It must be remembered that it is frequently a great physical effort to get the pus out, and what the patient may need most of all is rest.

The place of the bronchoscope in the treatment of lung abscess is not yet definitely established. It is of the greatest value where a foreign body or bronchostenosis due to some other cause is present. Obstruction due to inspissated secretion can readily be relieved. In cases of inoperable bronchiogenic carcinoma with associated abscess it is justifiable to employ bronchoscopic drainage of the abscess because of the distressing symptomatology. The instrument may be passed as often as two or three times a week without undue inflammatory reaction of the bronchial mucosa.[34]

Pneumothorax has been practically discarded in the treatment of abscess because of the danger of producing empyema, and because it has generally been disappointing as a means of closing cavities. It is not nearly so effective in this respect as it is in tuberculosis, and more than 50 per cent of cases require further surgery.[1] It is indicated only in cases where the cavity is adequately drained centrally by a bronchus, yet does not collapse satisfactorily. If compression therapy is attempted when adequate drainage has not been established, the patient may rapidly drown in his own pus. Also, it must be remembered that the cough is the body's most efficient means of expelling undesirable material from the upper respiratory tract, and anything which interferes with intrathoracic compression, and therefore with coughing, handicaps drainage.[13]

Arsphenamine and related compounds appear to be of value only in cases in which there is a copious growth of spirochetes, and even here the results are not very striking. It seems probable that small intravenous doses of neoarsphenamine regularly repeated may prevent the loss of lung tissue due to secondary spirochetal invasion, but is doubtful if it has any effect in healing the abscess itself.[22, 33, 37]

Nammack and Tiber[29] have reported the cure of 16 of 20 selected cases treated intravenously with guaiacol. Guaiacol, creosote and related compounds have long been known to be of some value in the treatment of the secondary invaders in pulmonary tuberculosis. When this material is injected intravenously with sodium iodide in a dilute alcoholic solution, it is recovered in the sputum or in the drainage from the operative wound during the first forty-eight to seventy-two hours. The patients in this series are reported to have felt better a short time after the administration of the drug was started, owing to subsidence of fever and cough and decrease in the daily output of foul sputum.

What are the indications for surgical intervention and what does the surgeon have to offer? In reporting cases from the Peter Bent Brigham Hospital, Cutler and Gross[9] state that the mortality could probably be lowered if all cases were submitted to early surgical drainage, but that many patients would then be operated on who would have recovered spontaneously. General opinion has held that six weeks to two months of active medical therapy should be tried, and this treatment prolonged if significant improvement occurs. Neuhof,[30, 31] in New York City, holds a more radical view, believing that abscesses should be operated on in the acute stage, that is in less than six weeks, if they are not progressing satisfactorily. He reports losing only 4 out of 45 consecutive cases operated on at the acute stage.

The surgeon has at his disposal various compression operations, one-stage or two-stage external drainage, and lobectomy and pneumonectomy. Phrenicectomy, extrapleural pneumolysis and thoracoplasty have all been used, but like artificial pneumothorax have been unsatisfactory and generally abandoned because of the danger of empyema and of a drowned lung resulting from collapse in the absence of adequate central drainage. These operations may be indicated in deeply situated abscesses communicating with a large bronchus, draining well and unlikely to rupture into the pleural cavity. However, such an abscess must be extremely rare, and is likely to heal spontaneously if central drainage is maintained.

Rib resection with external drainage is indicated when the abscess is at the periphery of the lung. Here the abscess drains poorly into the bronchi and is likely to rupture into the pleural cavity. There is little normal lung over the abscess, so that the danger of pneumonitis and hemorrhage from operation is minimal. A one-stage operation is indicated when there are well-formed overlying pleural adhesions. Neuhof[30] believes that with any peripheral abscess agglutinating adhesions of the two pleural surfaces occur very early, usually within two weeks, and that the important thing then is to find them. He claims to have found overlying adhesions in every one of his cases no matter how early he operated, though not always where he first looked for them. Most surgeons, however, have thought that during the early stages of the disease, while adhesions may be present, they do not entirely obliterate the pleural cavity, so that a one-stage procedure is not suitable. When adequate adhesions are not present a two-stage operation is in-

dicated, the first stage of which is done in order to create such adhesions by sewing the pleural layers together, packing gauze against the pleura or by some other means. The second stage, then, usually performed by cautery excision, consists in unroofing the abscess cavity and packing it thoroughly. Bronchial fistulas are always seen at operation, and in the subsequent treatment should be allowed to close only after all evidence of anaerobic infection is gone and infiltration is no longer present as shown by x-ray, a matter usually of at least six to eight weeks, and often some months.

A ruptured abscess with pyopneumothorax demands immediate drainage, and the prognosis in that event is usually graver than otherwise. Diagnostic or therapeutic needling of the lung abscess should be avoided because of the possibility of contamination of nonadherent pleural surfaces.

Lobectomy and total pneumonectomy are the newest weapons of the thoracic surgeon. These radical procedures are not well suited to acute suppurative lesions, because the presence of active infection makes bronchopleural fistula, empyema and other complications common. In carefully selected cases, however, one of these methods can be used, and indications for this type of surgery are being rapidly extended.

It is not possible at present to tell which cases of lung abscess are proceeding on to chronic pulmonary suppuration and gangrene.[25] The cases at the Children's Hospital were examined with this in mind, but aside from the time factor nothing significant was apparent. If healing by conservative means is going to occur at all, it seems to do so in the first six weeks. Abscesses which fail to resolve within this time probably will never do so, and the early use of radical surgery may then be indicated. Any correlation between the bacteriology of the sputum first obtained and the future course of the disease was not discernible because of insufficient bacteriological data. It was impossible in the postoperative cases to find out what sort of condition the child was in at the time of the operation, since it was performed in another hospital or at home. Postoperative lung abscesses are rarely treated in the same clinic in which the original operation was performed.

The etiology of the abscess in these cases apparently had no effect on the ultimate outcome. About one third of them resolved spontaneously, one third were improved or cured by surgical procedures and one third resulted in death, either from acute overwhelming sepsis or from chronic suppuration which did not respond to surgery.

PREVENTION

Can anything be done to lower the incidence of lung abscess? The measures of recognized importance in the prevention of all postoperative pulmonary complications should be rigidly observed. These are, essentially, reduction of trauma to a minimum, careful hemostasis, careful control of sepsis and, most important of all, extreme caution in operating on patients with any predisposition to atelectasis or formation of mucus.[10] To these we might add certain measures of probable significance in lessening the aspiration of infected material. Oral and dental hygiene should receive exact evaluation, and gingivodental infections be treated before any elective surgery is undertaken. Tonsillectomy and other operations on the upper respiratory tract must be considered as major surgical procedures and should be performed in a hospital. This is especially true in adults, where tonsillectomy should be performed only with definite indications. Teeth should be extracted in several sessions and where possible under local anesthesia. Interference with lung ventilation during and after operation should be closely watched for, and atelectasis resulting from mucous plugs relieved immediately by aspiration. A self-evident prophylactic measure is the adequate early treatment of pneumonia and other severe upper respiratory infections.

SUMMARY AND CONCLUSIONS

The majority of cases of nontuberculous abscess of the lung follow operative procedures of the upper respiratory tract or develop subsequent to unresolved pneumonia or other severe upper respiratory infections.

A primary lesion in the lung is apparently present before secondary infection with mouth organisms takes place; this may be infarction as a result of embolism or atelectasis as a result of bronchostenosis or bronchiolar obstruction.

There is no single bacteriologic etiology. The flora of the full-blown abscess is usually complex, and the quantitative importance and possible synergism of the various organisms are not at present understood.

It appears that oral infection is disproportionately common in adult patients with lung abscess of unknown etiology. This type of abscess is rare in infants and children.

The diagnosis of lung abscess is usually readily made from the history and general clinical picture, aided by roentgenography and bronchoscopy. The physical findings in the chest may be variable and inconclusive.

Accurate diagnostic localization is the most im-

portant prerequisite to both medical and surgical treatment.

Twenty to 30 per cent of cases heal with only supportive treatment, and all cases should receive an adequate trial of medical therapy, of which the most important feature is supervised postural drainage.

Collapse therapy, either by pneumothorax or by more extensive operative procedures, should never be attempted unless the abscess cavity has been adequately drained.

In peripheral abscess with reaction of the overlying pleura, external drainage done earlier than heretofore generally practiced, and based on accurate localization of the lesion, would seem to be indicated.

In abscesses which fail to respond to active medical therapy in two or three months, especially in children whose general condition is good, such radical surgery as lobectomy deserves early serious consideration, in order to avoid progression to chronic pulmonary suppuration and gangrene.

The prophylaxis of lung abscess includes careful operative technic, caution in operating on patients with any predisposition to atelectasis, close observance of lung ventilation during and after operation, conservative surgery on the upper respiratory tract with the use, if possible, of local anesthesia, improved oral hygiene and adequate therapy of respiratory infection in the early stages.

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REPORT ON MEDICAL PROGRESS

THE LEUKEMIAS*

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LEUKEMIA may be defined as an acute or chronic systemic disease involving primarily the blood-forming organs, characterized by a widespread, disorderly and profitless proliferation of the leukocytes and their precursors, manifest by the presence, often in very large numbers, of immature or abnormal white cells in the peripheral blood stream, and leading, at least in the vast majority of cases, to death within a comparatively short time.

CLASSIFICATION

From a clinical viewpoint, there is a rather sharp distinction between acute and chronic leukemia, and some authorities believe that there may be a fundamental difference in their essential nature and etiology. It is usual to classify the leukemias according to the particular type of white cell involved. We have, therefore, myelogenous, lymphatic and monocytic leukemia. The latter[1, 2] is almost always acute, and in the opinion of some investigators constitutes the majority of the cases of acute leukemia in adults. In addition, there have been described eosinophilic[3] and plasma-cell[4] leukemia. For most practical purposes it is probably sufficient to disregard the latter two forms, which at best are extremely rare, and to group all types of acute leukemia together as one clinical entity. It is advantageous, however, to retain a sharp distinction between chronic lymphatic and chronic myelogenous leukemia.

GENERAL OBSERVATIONS

Fortunately, leukemia is a rare disease. It is difficult to estimate its frequency with any degree of accuracy. In 8693 autopsies at the Boston City Hospital from 1920 to 1939 the condition in one form or another was encountered 78 times. During the same period a clinical diagnosis was made 333 times among 324,785 admissions to the medical and surgical wards. These figures closely approximate those of Ordway and Gorham,[5] who estimate that 1 case of leukemia occurs in every 1000 general hospital admissions. It must be remembered, however, that not a few patients with leukemia are nowadays referred by their physicians directly to hospitals whose staffs are particularly interested in the condition and which are especially equipped to treat the disease. Such cases are, therefore, not frequently seen on the wards of a general hospital.

All types of leukemia are much commoner in men than in women. This is especially true of chronic lymphatic leukemia.[6]

Acute leukemia is commonest in the early decades, a major peak being reached in the first five years of life, and a secondary though still significant rise occurring early in the second decade. While the disease may be seen at any time of life, it is distinctly rare after the age of fifty.

Chronic myelogenous leukemia is most commonly encountered between the ages of twenty and sixty, and reaches its greatest incidence between the ages of twenty-five and thirty-five, whereas chronic lymphatic leukemia is most often seen in persons between forty-five and sixty years of age. Any form of chronic leukemia is rare under the age of ten.

The etiology of leukemia is unknown, though it is probable that the chronic forms are closely related to the true neoplastic diseases, and some competent observers believe that the acute forms, while perhaps also of malignant nature, are more intimately related to infection.[7] Furth and his associates,[8] on the basis of extensive studies of experimental leukemia in mice, have concluded that all types of leukemia are true neoplastic diseases.

There are certain changes in the bodily economy common to all types of leukemia, and these should be referred to briefly. In all, the basal metabolic rate may be considerably increased[9] thus giving rise to certain signs and symptoms such as tachycardia, increased sweating, loss of weight, nervousness and easy fatigability, all reminiscent of hyperthyroidism. The basal metabolic rate may thus be of some value in the differential diagnosis of leukemia and the leukemoid states, and may further be of value in determining the need for therapy and the results thereof. The nitrogen balance varies within wide limits, but

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particularly in acute leukemia it is liable to be negative and the serum proteins tend to be low. These facts should be borne in mind when considering the therapy of the disease; the diet should at all times contain an adequate amount of protein for the individual needs, and it should be remembered that often large amounts of protein and nucleoprotein are lost to the body each day.

Anemia of the normocytic and normochromic type is an almost invariable accompaniment of the disease sooner or later. This anemia is chiefly a myelophthisic one, due to the overcrowding of the marrow by the exuberant growth of leukocytes, but in certain cases it may be greatly enhanced by hemorrhage, particularly into the gastrointestinal tract. The presence or absence of anemia is of great diagnostic value in the acute forms, and in both acute and chronic leukemia, progressive anemia may bring about symptoms of major importance and call for specific and energetic therapy. The presence of severe anemia usually indicates an early fatal outcome, especially in the acute and the chronic lymphatic types.[6]

Any form of leukemia, but more especially the acute, may be accompanied by spontaneous hemorrhages of minor or major clinical importance. In the acute forms, this hemorrhagic state is dependent to a great extent on the frequent paucity of blood platelets; in the chronic leukemias the same may be true, but there is the added factor, not manifest in the peripheral blood stream, of direct damage to the finer blood vessels by the invading leukocytes.

It is of the utmost importance to recognize the fact that any organ or structure of the body may be involved by the leukemic process, for thus are the extremely protean signs and symptoms of the disease brought about. It is perhaps germane to the present purpose to draw attention to lesions of five systems in particular, for lack of recognition of the fact that these systems are frequently involved may lead to failure to recognize the fundamental systemic disorder, or failure to treat as leukemic manifestations symptoms or signs easily attributable to other causes.

In the mouth and pharynx, there may be ulcerations, necrosis, bleeding and even noma, especially in the acute forms of the disease. Monocytic leukemia in particular is associated with severe oral lesions.[2] It is not unusual to find in acute leukemia that the first symptom is unexpected bleeding following a minor operation such as a simple tooth extraction. More than one patient with leukemia has first consulted his dentist for a supposedly local sign or symptom, and I know of one oral surgeon who very wisely always does a complete blood examination in any puzzling or peculiar "mouth case." It should further be borne in mind that in elderly people a sore throat of unusually long duration or enlargement of the tonsils without obvious infection may be due to lymphatic leukemia.

The eye is involved sooner or later in over half the cases of leukemia,[10] though obvious clinical manifestations may not be present. Retinitis, retinal hemorrhages, edema of the disks and marked engorgement of the vessels are the commonest changes noted. Diminution of visual acuity is not rare, and sudden blindness may be the first symptom noted. Exophthalmos, either unilateral or bilateral, may occur, especially in children.

The central nervous system is similarly often involved,[11] lesions of the cranial nerves, reflex changes and hemorrhages, large or small, being the most commonly seen. In rare cases, meningitis is simulated and in the sterile spinal fluid are seen the immature cells characteristic of the individual type of leukemia at hand.

In the skin, a bewildering variety of lesions occurs.[12] Indeed in certain cases the chief, if not the only, clinical manifestation is a dermatologic one. There may be single or multiple nodules, often of a quite nonspecific character. Or there may be, particularly in chronic lymphatic leukemia, a diffuse reddening and scaling of the skin accompanied by intense itching. As in the non-leukemic lymphomas, herpes zoster is not uncommon, and in rare cases it may be of universal distribution.

Changes in the bones or symptoms related thereto are common and important, particularly in the acute leukemia of children, in whom rheumatic fever may be closely simulated.[13, 14] The fact that children with leukemia often present a peripheral blood picture which is atypical makes this fact particularly significant, for the joint symptoms may so dominate the clinical picture that the true diagnosis is completely overlooked.[15] Periosteal infiltrations and a moth-eaten mottling of the ends of the long bones as shown in the roentgenogram are particularly suggestive of a leukemic process.

It is thus obvious that the clinical manifestations of leukemia are most varied and that the presenting signs and symptoms may not necessarily suggest the widespread nature of this uniformly fatal disease, which may at first manifest itself as a comparatively isolated lesion of a system which is only remotely connected with the blood-forming organs, and which not infrequently falls within the province of the specialist rather than that of the general internist.

As has been said above, for most practical pur-

poses all types of acute leukemia, whether lymphocytic, monocytic or myelogenous, may be regarded as one and the same disease. This is so because the prognosis and treatment in all are essentially the same. For those particularly interested in the more exact and definitive classification of the acute leukemias, we refer to the studies of Forkner.[2]

ACUTE LEUKEMIA

The onset of acute leukemia is usually insidious, more rarely abrupt. It should be constantly borne in mind that the disease is essentially one of infancy and childhood, though no age is spared. Often an ill-defined and gradually increasing weakness or an unexplained torpor and lassitude constitute the presenting symptoms. Equally common, and equally deceiving to the unwary, are symptoms of sore throat, ulcerations of the buccal mucosa or hemorrhages following some minor surgical procedure. Only too often, the localized nature of the symptoms overshadows the systemic nature of the disease. Peirce[16] has drawn attention to the fact that in many cases the first symptoms seem to be related to upper respiratory infection from which the child does not recover as it would seem he should. A gradually increasing pallor may be the sole sign apparent. Nausea, vomiting, abdominal pain, melena and anorexia are not infrequent. Occasionally these may be of such character as to suggest an acute abdominal emergency, and patients have been operated on for supposed appendicitis or acute intestinal obstruction.[15] Fever, often of a septic type, is usually present, and may be seen even when the patient appears to be in good general condition.

Early in the course of acute leukemia the physical examination may be essentially normal. Sooner or later, however, there is usually generalized enlargement of the lymph nodes and the spleen is commonly palpable on deep inspiration. Hemorrhages into the skin, mucous membranes and eyegrounds are common. The frequent oral lesions have already been discussed. Occasionally — and these cases may be most deceptive — acute leukemia in children masquerades as rheumatic fever.[15] It is most important to remember that under a variety of signs and symptoms — many of them apparently of minor importance — may lurk one of the most dreaded of all children's diseases.

The peripheral blood in the majority of cases shows a characteristic picture. The white-cell count may be normal, moderately elevated or markedly depressed. Leukopenia is particularly prone to be found in the early stages of the disease. Only rarely does one encounter the great increase in white-cell count so frequently seen in the chronic leukemias, and it cannot be emphasized too often that the total white-cell count per se has little or nothing to do with the diagnosis.

In the classic case, there is a preponderance of very immature white cells of one or another series. Often stem cells, characterized by their deep blue cytoplasm and their prominent nucleoli within the nucleus, predominate in the blood smear. If 90 per cent of the white cells — no matter what the total count and no matter what the symptoms — are true stem cells the diagnosis is almost certainly acute leukemia. But an occasional stem cell may be found, especially in children, in a large variety of conditions, and one must always be cautious before making a final diagnosis. Relatively mature white cells of any series may compose the majority of the white cells in the blood smear, and rarely there are but few very young white cells present. Under the latter circumstances it is most difficult to diagnose acute leukemia with any assurance. Occasionally one finds the so-called hematologic hiatus, in which there are present in considerable numbers both extremely immature and quite mature white blood cells. No intermediate forms are present. Under these circumstances a diagnosis of acute leukemia is almost certain.

The platelets in acute leukemia are almost invariably greatly reduced in number, and this fact is of the highest importance in differentiating this disease and certain cases of overwhelming infection in which as a rule the platelets are increased in number, often markedly so.

Sooner or later in acute leukemia a moderate or severe anemia develops. It must be remembered, however, that the life of the red cell in the peripheral blood is such that anemia arising primarily from bone-marrow failure does not take place for a matter of weeks, so that the absence of anemia in the presence of other unequivocal signs and symptoms of leukemia should not be construed as evidence against such a diagnosis if the case be seen early.

Thus progressive anemia, a preponderance of very immature white cells in the blood smear and thrombocytopenia usually indicate the presence of acute leukemia. If there be in addition splenomegaly and slight generalized lymphadenopathy, the diagnosis becomes practically certain. It is important to remember, however, how protean may be the clinical manifestations of the disease and how variable the blood picture.

Acute leukemia must be differentiated chiefly from infectious mononucleosis, aplastic anemia, agranulocytic angina and overwhelming sepsis.

Infectious mononucleosis may usually be distinguished by the presence of the characteristic white

cells of this disease,[17, 18] by the absence of anemia and thrombocytopenia and by the presence of a positive sheep-cell agglutination test.[17] This test is never positive in leukemia, and if positive in dilutions of 1:64 or greater is clear evidence that one is dealing with infectious mononucleosis, unless there has been a recent administration of horse serum.

In aplastic anemia there are few if any really immature white blood cells in the peripheral smear, and classically all three essential formed elements of the blood are concomitantly diminished. The basal metabolic rate is not increased, and there is rarely splenomegaly and never generalized lymphadenopathy of any moment. Yet in certain cases it may be impossible without bone-marrow biopsy to be confident of the diagnosis.

Cases of acute leukemia have often been erroneously diagnosed as agranulocytic angina.[19] In view of the different prognosis and treatment of the two conditions, it is of the utmost importance to differentiate them clearly. In general it may be said that, in agranulocytic angina, anemia and thrombocytopenia are absent, that extreme leukopenia is the rule, and that one rarely sees immature white blood cells in any number during the acute stage of the disease. During recovery, however, the white-cell count may occasionally rise to 20,000 or even 100,000, and there may be many myelocytes and even younger white cells in the blood. Under these circumstances it may be difficult to say whether one is dealing with a marrow overactive following a period of pathologic inactivity or with leukemia. Agranulocytic angina is very rare in children, but to make matters more difficult, what appears to be the true disease has been followed after weeks or months of remission by true acute leukemia. The majority of these cases have been those of children or very young adults, so that one should always be most cautious in regard to prognosis of the leukopenic state in this age group.

Overwhelming sepsis may manifest itself by anemia, and by the presence in the blood of extremely immature white cells, often in considerable number. Usually, however, the platelets are increased rather than decreased in number and, in the majority of cases, the infection has obviously preceded the development of the abnormal blood picture, so that the differential diagnosis is clear. But when one remembers that sepsis is a common complication of acute leukemia, one realizes the difficulty of making an accurate diagnosis in any given case. When in doubt, it is probably wise to treat the case as one of infection and give a guarded prognosis.

The course of acute leukemia is almost invariably progressive onward to death within a few weeks or months. Very rarely there may be remissions of considerable duration, during which the patient returns to apparent health and the blood picture to normal.[20] The physician must be careful not to allow such temporary improvement to lull him into a false sense of security.

There is no form of treatment known that affects the course of acute leukemia. X-ray therapy is seldom of even temporary advantage, and it may cause severe reactions. If the diagnosis of acute leukemia is clear, radiation is probably contraindicated. If there is an anemia, causing untoward symptoms, transfusions of blood may be given, but severe reactions are liable to take place, even with correctly matched bloods. One of the most difficult tasks which confront the physician is that of withholding any treatment other than symptomatic in these cases. Yet such is usually the wisest course, and the best that one may hope for is that the end will come quickly or that one's diagnosis is incorrect.

CHRONIC MYELOGENOUS LEUKEMIA

The onset of this disease is usually insidious, and the condition has most often been in progress for many months by the time the patient seeks medical advice. The commonest symptoms are loss of weight and strength, increased sweating, weakness and distention of the abdomen. Often there is generalized abdominal discomfort, especially after meals, owing to the presence of a greatly enlarged spleen which encroaches on and interferes with the gastrointestinal tract. More rarely there may be acute pain in the splenic region attendant upon the very common splenic infarcts. Pallor, dyspnea, nausea and anorexia are common. Occasionally there may be a marked hemorrhagic tendency. Eye symptoms are not unusual, and may be due to retinal exudate, hemorrhages or edema of the disks.

When the patient is first seen, the spleen is usually enlarged, often greatly so; but early in the disease, splenomegaly may not be a prominent feature and its absence does not exclude a diagnosis of leukemia. Moderate hepatomegaly is usually present. Generalized lymphadenopathy is rare, but occasionally enlarged lymph nodes are found in the neck, axilla and elsewhere.

The total white count is classically elevated—often to a very great degree. White counts of 200,000 to 500,000 are not unusual and counts well over a million have been reported. It must, however, be constantly borne in mind that the total white count per se is not the most important fac-

tor in the diagnosis. One may find white counts of 150,000 and over in patients who do not have leukemia and patients with leukemia may show a marked lowering of the total white count. The important feature, in so far as the blood is concerned, is the increased percentage of very immature cells. In the average case, myelocytes in various stages of development comprise the majority of the cells, varying from 10 to 80 per cent. Stem cells are seen in the more rapidly advancing types, but very rarely to the degree seen in acute leukemia. It is important to bear in mind that marked fluctuations in the total white-blood-cell count and in the differential may occur without any treatment whatever and that these variations are of little or no prognostic value.

Sooner or later a normocytic, normochromic anemia develops and the red cells often show a polychromatophilia and a considerable number of nucleated red cells. The platelets may be normal, increased or decreased in number. A marked decrease is of grave prognostic import.

The usual case with splenomegaly, greatly increased white-cell count and immature granulocytes can be diagnosed with some assurance, but it is important to recognize that a wide variety of conditions may give rise to a leukemoid blood picture.[15, 21] Space does not permit an analysis of the differential points involved, but it should be mentioned that blood pictures simulating myelogenous leukemia may be seen in sepsis, metastatic carcinoma, miliary tuberculosis, sulfanilamide poisoning, polycythemia vera and especially in agnogenic myeloid metaplasia.[22] In this last condition, which may very closely simulate myelogenous leukemia, particularly in the aleukemic phase, x-ray treatment should be given with the greatest caution if at all, for the chief seat of blood formation in these cases is the spleen. It is obvious that if by radiation one destroys even in part the sole or major source of blood formation, one may do more harm than good.[22, 23]

The disease is usually steadily progressive and leads to death in two to four years. Some 10 per cent of patients survive ten years or more from onset. In the majority of cases the course of the condition is such that the experienced physician can foretell with some accuracy when a fatal termination is near, but it should be pointed out that sudden and unexpected death (usually from cerebral or gastrointestinal hemorrhage) occasionally occurs, and the family should be apprised of this fact.[15]

To date, the most effective treatment is irradiation of the spleen. It is best to leave the exact amount of irradiation given to the radiologist in charge, but certain generalities may properly be pointed out. As a rule, one gives to the spleen approximately 600 r at 250 kilovolts in divided doses over a period of a week to ten days, watching the white-cell and differential counts carefully during the treatment. The white-cell count should not be allowed to fall below 15,000 or 20,000 (when it has previously been greatly elevated). If the initial counts are already relatively low, x-ray treatment should be undertaken with caution. The beneficial results in so far as the patient's general sense of well-being is concerned often do not take place for a matter of several weeks, and this fact should be drawn to the patient's attention before therapy is instituted. If the initial red-cell count is below 3,500,000, one or more blood transfusions are indicated before irradiation therapy is undertaken. If the platelets are few, x-ray therapy should be undertaken with caution. It should be pointed out, however, that appropriate radiation therapy in cases of myelogenous leukemia not infrequently results in an increase of platelets when these elements have previously been few in number.

Further treatment, after the first course, may be withheld until such time as the patient's symptoms appear to call for alleviation. It is well to remember that it is problematical whether life is prolonged by irradiation, and that our efforts should be aimed chiefly at the alleviation of symptoms rather than the mere reduction in the total white-cell count.

If x-ray therapy is not available, — and at present it is available only in the large centers, — one may advantageously use Fowler's solution by mouth. For the details of such treatment, the reader is referred to the article by Forkner.[2] In general, it may be said that from 5 to 40 minims of Fowler's solution are given three times a day until the desired lowering of the white-cell count has taken place, and the patient is then continued, with rest periods, on a small maintenance dose. It is important to recognize, however, that treatment by arsenic must be carefully suited to the individual case, and in all instances the dosage must be very gradually stepped up from the minimum to the optimum. Unfortunately, while it is true that marked improvement in the blood picture can usually be brought about by this form of treatment, the fact remains that toxic manifestations such as anorexia, skin lesions, diarrhea and peripheral neuritis are not uncommon, and they may render the patient even more uncomfortable and ill than does the fundamental disease, so that x-ray therapy remains the treatment of choice.

In our enthusiasm for x-ray therapy and our satisfaction with the results obtained, we should not lose sight of the advisability and indeed the necessity of general supportive measures. An adequate diet, plentiful in vitamins and proteins, should be maintained. Proper rest periods during the day may enable a patient to carry on his activities with reasonable success. Iron in the form of ferrous sulfate is frequently helpful. Transfusions of blood — often repeated many times — are of great advantage if there be a marked anemia.

CHRONIC LYMPHATIC LEUKEMIA

There is a close relation between lymphatic leukemia and lymphosarcoma. The latter condition may be widespread and reach massive proportions without there being any manifestations of the condition in the peripheral blood, but not infrequently a patient with an isolated lymphosarcoma will, with the passage of time, develop the peripheral blood picture of classic lymphatic leukemia, and conversely there may arise in patients with lymphatic leukemia large tumor masses in the mediastinum or elsewhere. For most practical purposes these two conditions may be regarded as identical.

Chronic lymphatic leukemia occurs in an older age group than does chronic myelogenous leukemia and is less amenable to treatment, but is more likely to run a long and relatively benign course.

Not infrequently the disease is for a long time symptomless. Each year in any large general hospital, cases with the disease, but with no symptoms attributable to it, are discovered on the medical or surgical wards solely through routine blood examinations. The commonest symptoms are weakness, pallor, general malaise, vague gastrointestinal symptoms, general enlargement of lymph nodes and a hemorrhagic tendency. In certain cases a diffuse reddening and itching of the skin is the presenting and indeed the only symptom. Both melena and hematuria are seen with sufficient frequency to merit particular attention.

On physical examination, one may find nothing of note, More usually, however, there is a widespread lymphadenopathy, the lymph nodes themselves being of a fairly uniform size and distribution and of the consistence of soft rubber. The spleen is usually enlarged, though only rarely does it reach the massive size attained in chronic myelogenous leukemia.

The white count may be normal, subnormal or — most commonly — greatly elevated. White-cell counts of over 500,000 are not unusual. The differential count shows an overwhelming preponderance of lymphocytes, the majority of which appear normal. In the more rapidly advancing cases it is usual to find a certain percentage of young lymphocytes, though their presence even in considerable quantities does not invariably indicate an early fatal outcome, as is witnessed by the fact that one patient — a sixty-five-year-old man — has had some 20 per cent of lymphoblasts in the peripheral blood stream for a period of six years but is still able to carry on an active and strenuous career. Anemia is an almost invariable accompaniment sooner or later. The platelets may be increased in number, but more commonly they are sharply decreased, and their paucity in this disease does not carry the same unfavorable prognosis that the similar finding in chronic myelogenous leukemia does.

In the typical case the diagnosis can hardly be missed, but when there is an associated leukopenia it may be almost impossible to make, and on occasion the disease must be distinguished from pertussis, infectious mononucleosis, aplastic anemia and rarely atypical tuberculosis.

On the average, patients with chronic lymphatic leukemia survive only three or four years from onset of the disease, but fully 10 per cent live ten years or more, and rarely the patient may survive an even longer time, and even then succumb to some other quite unrelated condition.[24] This fact should be borne in mind, particularly in elderly people with few or no symptoms referable to the leukemic picture.

As with chronic myelogenous leukemia, irradiation of the spleen or chest, preceded by blood transfusions if there is a co-existing anemia of moment, is the treatment of choice. It is very questionable, however, whether there is any advantage in treating those patients who are symptomless. The details of the x-ray therapy should be left to the radiologist in charge, but the same general principles hold as with chronic myelogenous leukemia.

In certain cases, Navitol in large doses — 50 or 75 drops a day — seems to be advantageous, particularly in alleviating itching in those patients who have generalized skin lesions. If tumor masses of lymphosarcoma rise and cause symptoms, irradiation should be given directly to them.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTEMORTEM AND POSTMORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

CASE 26011

PRESENTATION OF CASE

A seventy-four-year-old Swedish widow was admitted to the hospital complaining of pain in the lateral side of the right knee of three and a half to four months' duration.

The patient was well until four months before admission when all but four of her teeth were removed because of "pyorrhea." The extractions were performed without known complication. About three and a half months before entry, without known trauma, she first felt a slight, transient, stabbing, non-radiating, non-throbbing pain in the lateral side of the right knee. The pain increased rapidly in severity and in duration so that about two months before admission she was forced to stop her daily housework. She went to bed for two weeks and applied liniments locally, but without improvement. She was able to work again for a few weeks, but was forced to stop when the pain on motion of the joint became so severe that at times she felt faint. However, there was no discomfort when the leg was at rest. The knee had been slightly swollen since the onset of her illness, but it increased rapidly in size during the three weeks before admission. She had been unable to walk for two weeks because of pain. She lost about 5 pounds in weight (normal weight 160 pounds) during the present illness.

Physical examination revealed a fairly well developed and nourished woman in no apparent acute distress. The examination was negative except for that of the right leg which showed a painful, swollen, questionably fluctuant, hot area located above and lateral to the knee. The overlying tissues were tense and relatively immobile.

The temperature was 99°F., the pulse 110, and the respirations 20.

Examination of the blood showed a red-cell count of 4,200,000 with 70 per cent hemoglobin (Tallqvist), and a white-cell count of 15,800 with 83 per cent polymorphonuclears. The corrected sedimentation rate was 1.2 mm. a minute. The urine was negative; there was no Bence-Jones protein. The blood Hinton test was negative. The serum calcium was 10.2 mg. per 100 cc., the phosphorus 4.3 mg., and the serum phosphatase 5.7 units. X-ray examination showed a localized area of bone destruction in the lower end of the right femur; it began at the anterior joint margin and extended upward 4.5 cm. The lesion was about 3 cm. in depth and ulcerated, with a 3-cm. soft-tissue mass. There was slight periosteal proliferation, more marked on the medial aspect of the femur slightly away from the area of destruction; this reaction simulated "collar formation." There was also some new-bone formation at the margins of the lesion, seen best in the anteroposterior view. The soft tissues were only slightly thickened. There was no fluid in the joint. The patella was elevated and slightly below its normal position. The left knee and other bones were normal. The chest was negative. An intravenous pyelogram was negative. A barium enema was negative.

On the fifteenth hospital day an operation was performed.

DIFFERENTIAL DIAGNOSIS

DR. THOMAS J. ANGLEM: The important fact in this case is that we have a destructive lesion in the lower end of the femur in a woman aged seventy-four, who gave a story of pain referred to the lateral aspect of the knee for three and a half to four months, gradually increasing in severity and associated with progressive swelling referred to that part. I think we should save considerable speculation and exploration of blind alleys by viewing the x-ray films.

DR. AUBREY O. HAMPTON: I cannot add anything to the description in the record. I can point out the things that were described. The lesion is here; it is almost a purely destructive process, which extends down to and underneath the cartilage of the knee. It does not destroy the cartilage but is exactly against it and forms an area of destruction somewhat similar to that of a giant-cell tumor. The remainder of the findings are not the least bit similar to those of a giant-cell tumor. The "collar formation" here is quite definite. We thought we could rule out an infectious process because there was no fluid in the joint.

DR. ANGLEM: The first problem is, as Dr. Hampton has already said, to rule out an infectious process. I think we might well attack the differential diagnosis by eliminating one possibility after another. The first paragraph provides material for a tempting hypothesis. The patient was said to have had extraction of all but four of her teeth for pyorrhea, and it is not unreasonable to assume that she may have had apical dental infection. The trauma incidental to dental ex-

traction might have opened up pockets and permitted passage of infective material into the blood stream and some of the infected emboli might have been transported to the femur and resulted in osteomyelitis. Certainly the clinical history and clinical findings are consistent with such a hypothesis, as also are the slight elevation of temperature and the elevated white-cell and polymorphonuclear counts. On the other hand osteomyelitis is extremely rare at seventy-four, and when it does occur it is more likely to develop in the shaft than in the end of the bone. Dr. Hampton has pointed out features in the x-ray picture that are difficult to explain on this basis: the absence of any evidence of joint involvement and the lack of fluid in the joint. If this were osteomyelitis, I think also that with a process as active as this we should expect to see more soft-tissue swelling in the film.

Could it be tuberculosis? Tuberculosis, again, is very uncommon at this age; it is essentially a disease of youth. On the other hand, it occurs characteristically in the ends of the long bones and typically involves the epiphysis, as this lesion does. I think the mode of onset of the disease in this case is distinctly against the diagnosis of tuberculosis. The rapidity with which the disease process developed, the severity of the pain and the character of the swelling associated with the pain are against the diagnosis of tuberculosis. The swelling referred to the knee, as described here, is localized to the lateral aspect of the knee, and the skin is described as tense and tender. A fluctuant sensation was elicited on palpation. These findings are not characteristic of what we see in tuberculosis. There the swelling is more apt to be fusiform and symmetrical rather than localized to one side; tenderness is not so pronounced; local heat is also unusual, and the skin is usually white. We are not told the color of the skin, but since there was considerable local heat there was presumably some erythema. Could this be syphilis or, more specifically, gumma of bone? Gumma of bone can produce an osteolytic lesion that must be considered in a differential diagnosis of this kind. I do not believe we need consider syphilis very seriously, however, since the Hinton test was negative. Again, the mode of onset of the disease, the rapidity with which the symptoms developed and the severity of the pain are all against the diagnosis of syphilis. So too are the local findings, the degree of swelling, the amount and extent of the tenderness, the boggy character of the swelling, the tenseness of the skin and the feeling of fluctuation.

There are certain data in the history which seem to me to be consistent with any of the possible diagnoses which I have mentioned here. But they are not diagnostic. The mild degree of anemia, the 5-pound weight loss, and the elevated sedimentation rate are not helpful in arriving at a diagnosis. The elevated sedimentation rate is consistent with a diagnosis of osteomyelitis or tuberculosis, but since I have no knowledge of the behavior of the sedimentation rate in cases with bone tumor, this finding does not help me.

If this is tumor, is it a metastatic tumor or a tumor primary in the bone? Metastatic tumor is much more consistent with this patient's age than is primary tumor. The tumors which commonly produce metastatic bone lesions are mammary, thyroid, renal and adrenal cell carcinomas. There is nothing in the history or physical examination to suggest the presence of any of these primary tumors. We have additional evidence against renal cell or adrenal cell carcinoma in the negative intravenous pyelogram; and the negative barium enema presumably rules out carcinoma of the lower gastrointestinal tract, which occasionally produces by metastasis an osteolytic tumor of bone. Furthermore, the x-ray picture is not at all typical of metastatic carcinoma. Metastatic lesions occur more often in the shaft than in the ends of the long bones and are apt to be centrally located. They are expansive to a certain extent and produce thinning of the cortex and perforation, but seldom widespread destruction, such as we have here, and rarely any appreciable invasion of soft tissue. Here we have definite soft-tissue invasion by the tumor. The phosphatase level is consistent with a metastatic tumor. The elevation of the serum phosphatase is further evidence against inflammatory lesions, but the elevation is so slight that we cannot attach much significance to it.

Can this be a metastatic lymphoblastoma? This is a little more difficult to rule out. The general appearance of the lesion is more consistent with that of a metastatic lymphoblastoma than it is with that of a carcinoma, except for one thing — in my experience I have not often seen soft-tissue invasion by metastatic lymphoblastoma. Furthermore, against the diagnosis is the fact that we seldom see metastatic disease from lymphoblastoma except in advanced cases, where the general disease is obvious. I think we can rule it out.

We have left then the primary bone tumors, and the one most likely to occur at this age is myeloma. This is not multiple myeloma, since we know that the x-ray study did not reveal any other lesion. Against the diagnosis of solitary myeloma, which is an extremely rare disease, particularly in women, is the character of the x-ray findings. The lesions in

plasma-cell myeloma are more apt to occur in the shaft and, like those of metastatic carcinoma, are somewhat expansile in character and produce thinning of the cortex, sometimes perforation, but very rarely extensive cortical destruction or soft-tissue invasion. Furthermore, there was no Bence-Jones protein in the urine. This is not conclusive, however, since the test may be negative in 50 per cent of cases. We are not told what the serum protein was.

Two or three years ago we should not have included in such a differential diagnosis the entity known as primary reticulum-cell sarcoma of the bone; but all of us now recognize the existence of such a tumor, and it must be considered. The clinical symptoms are entirely consistent with the diagnosis. I know, however, of no case that has occurred at this advanced age. But, cases have been reported in the fifties, and we cannot rule it out on that basis. The x-ray findings are consistent with the diagnosis. I do not believe I can rule it out with any measure of certainty.

We have left, then, osteogenic sarcoma, which is extremely uncommon at this age. Furthermore, osteogenic sarcoma very rarely invades the epiphysis, and there are other features in the story which are seemingly inconsistent with the diagnosis. For example, we are told the pain was relieved by rest, whereas the typical story is that the pain is unrelieved by rest. We cannot rule it out on that basis because there are cases in which rest does result in the relief of pain. In spite of these objections we have a strong case for osteogenic sarcoma of the osteolytic variety. Although osteogenic sarcoma does not occur commonly in adults, when it does, it is very often of the osteolytic type. Also perhaps of significance in this case is the fact that the osteolytic type of tumor has a predilection for the femur and is particularly prone to occur in the outer condyle. The x-ray findings, I think, are entirely consistent with the diagnosis of osteogenic sarcoma. One factor that disturbs me more than any other is one point that Dr. Hampton brought out; namely, that the disease process extended down to the joint cartilage. In osteogenic sarcoma which has invaded the epiphysis there is usually a layer of intact spongiosa between the area of destruction and the joint cartilage. In spite of that point I think it is osteolytic osteogenic sarcoma, and in further support of that diagnosis is the fact that there was a serum phosphatase of 5.7 units.

Dr. Fuller Albright: The serum phosphatase is expressed in Bodanski, not Kay, units. The normal level is from 2 to 5 units, but the reading is still a little high.

Dr. Anglem: Then I do not get much support from it, and shall have to retract that statement. However, in cases with osteolytic osteogenic sarcoma the phosphatase need not be elevated very much and may even be normal. I shall stick to my diagnosis of osteolytic osteogenic sarcoma.

Dr. Ernest M. Daland: How do you rule out a benign giant-cell tumor?

Dr. Anglem: I do not believe the x-ray picture has any of the characteristic features of a giant-cell tumor: it is not expansile; there is no evidence of trabeculation; there are destruction at the cortex and invasion of the soft tissue.

Clinical Diagnosis

Osteogenic sarcoma.

Dr. Anglem's Diagnosis

Osteolytic osteogenic sarcoma.

Anatomical Diagnosis

Osteogenic sarcoma of femur.

Pathological Discussion

Dr. Tracy B. Mallory: A punch biopsy was taken on this woman, and we further complicated the picture by reporting it was consistent with a benign giant-cell tumor. The amount of destruction of the bone was so great, however, that the surgeons were of the opinion that amputation was necessary regardless of the diagnosis and it was carried out. On the final specimen we reversed ourselves and called the lesion an osteogenic sarcoma. It was a peculiar looking one, however, and I am not altogether happy with the histological diagnosis. About three quarters of the specimen looked like an organized hematoma. There were areas showing definite osteoid formation by tumor cells, however, and there were a few areas that really looked sarcomatous.

Dr. Hampton: I understood you thought that this might have developed on a giant-cell tumor.

Dr. Mallory: It seems as though something of that sort must be assumed, because it is not in any respect a typical osteogenic sarcoma.

Dr. Daland: I operated on this patient with the biopsy diagnosis of benign giant-cell tumor, although we did not believe it was at all definite. Because the limb was useless and painful and because this woman was seventy-four we decided that amputation was the best method of handling the situation, and prepared to amputate between tourniquets. We cut down on the tumor, making a transverse incision above the patella. We came to a large mass which was mostly blood clot and organized hematoma. It was difficult to deter-

mine the exact nature of the tumor. The entire knee joint was full of this material. We were much impressed with the lining of the cavity; it was not the smooth lining that you see in benign giant-cell tumor, and the depth of the cavity was much greater than that indicated by the x-ray film. For that reason we did not accept the diagnosis of giant-cell tumor and continued with the amputation.

CASE 26012

Presentation of Case

A twenty-five-year-old Negro, born on Cape Verde Islands, was admitted to the hospital complaining of pain in the left upper arm.

Six years before admission the patient noticed a moderately severe, non-radiating, aching pain in the middle of his left upper arm. The pain responded favorably to "liniment" and massage, and he was asymptomatic after two or three days. Since then he had suffered similar yearly recurrences, without swelling, local heat, radiation of pain, chills or fever. Between attacks he felt well and was able to work as a picker in a Cape Cod cranberry bog. Six months before entry, however, the pain became much more severe and involved the left elbow, and he was unable to use the arm for two to three weeks. Since then he had experienced almost monthly attacks of pain in either the elbow or middle arm, and for two months before entry, the discomfort had been constant. Five days before admission the left arm and elbow became swollen, hot and very painful. He was unable to move the elbow joint and held it in a semiflexed position in a sling. There were no other symptoms.

Physical examination revealed a well-developed and well-nourished man in no acute distress. The examination was entirely negative except for the left arm. The elbow was held in semiflexion; the upper arm was hot, puffy, edematous, but nonfluctuant. There were no palpable axillary lymph nodes. The arm was protected by the patient and held in semiflexion. The blood pressure was 130 systolic, 70 diastolic.

The temperature was 98°F., the pulse 80, and the respirations 20.

Examination of the urine was negative. The blood showed a red-cell count of 3,600,000 with 80 per cent hemoglobin (Sahli), and a white-cell count of 12,200 with 81 per cent polymorphonuclears; the blood smear was negative. The tuberculin test, using 0.1 cc. of a 1:10,000 dilution, was negative. Two blood Hinton tests were positive, the Wassermann test negative. The blood nonprotein nitrogen and serum protein determinations were normal. The spinal-fluid Wassermann and gold-sol tests were negative; the spinal-fluid protein was 14 mg. per 100 cc.

Roentgenograms of the left humerus showed a loculated cavity involving the medulla of the mid-third of the shaft of the humerus about 13 cm. in length. The cavity was fairly well defined in its lower end but poorly defined in its upper end where there was apparently some destruction in the adjacent bone. The cortex was destroyed

Figure 1. *Left Humerus the Day of Operation.*

in at least three places and was thinned in others. There was marked rough periosteal new-bone formation over the lesion, as well as over the area of destruction at its upper margin. There was soft-tissue swelling about the lesion, and this appeared to be due to edema. An area of increased bone density lay close to the lower edge of the lesion. There was no evidence of pulmonary lesions. Films of the pelvis, bones of the hands, femora, tibia, knee joints and skull showed no abnormalities. Intravenous pyelograms showed several areas of calcification overlying the upper pole of the left kidney, and apparently outside it.

The patient ran an irregular, spiking type of temperature, ranging from 98.6 to 103°F. An x-ray film (Fig. 1) taken about two weeks later, on the day of operation, showed essentially the same changes.

Differential Diagnosis

Dr. Fuller Albright: I do not know much about bone disease of this type. May I see the x-ray films?

Dr. Aubrey O. Hampton: I know the answer so I prefer that you ask questions.

Dr. Albright: The periosteal proliferation suggests syphilis. We notice that the epiphysis of the bone is not involved. In other words, it is a midshaft process. I do not believe the calcification over the kidney can possibly be in the adrenal gland. Is that correct?

Dr. Hampton: Yes; it looks like a tuberculous node.

Dr. Albright: I have no other questions. Have you any other comment not brought out by the description?

Dr. Hampton: No.

Dr. Albright: What does one consider? The first possibility is tumor. The facts that there was no soft-tissue mass and that the lesion had lasted six years are against tumor, especially a Ewing tumor. There is no evidence of another type of tumor; hence, tumor is unlikely. Secondly, one should think of infection. In the beginning the absence of fever was against infection; however, at the time of admission the fever and white count were strongly in its favor. The various types to consider are tuberculosis, syphilis and chronic osteomyelitis. Tuberculosis does not usually start at the midshaft. The pain is somewhat against syphilis, but the positive Hinton test and the periosteal proliferation are in its favor. It is most unusual not to have a positive Wassermann test in active syphilis. I do not believe he had bone syphilis, but it remains a distinct possibility. The symptoms of chronic pyogenic osteomyelitis can be very obscure, and these areas of increased density around the bone certainly look like those of osteomyelitis. The facts are consistent with such a diagnosis, and I do not believe it can be ruled out.

Dr. Hampton: What do you think of the sharp cystic appearance of the areas of rarefaction?

Dr. Albright: It fades out up here, but, as you say, is sharp on this side. There is no sequestrum, which, if present, would favor osteomyelitis, but I do not believe that sequestration necessarily occurs in osteomyelitis. This diagnosis cannot be ruled out and is my best bet.

The third possibility is bone cyst. However, the general appearance of the lesion is not like that of a bone cyst, and I do not believe it is going to turn out to be that. Why I say this, I do not know, other than because it does not look like it to me.

Dr. Hampton: How about the periosteitis?

Dr. Albright: Thank you; the presence of periosteitis is strong evidence against bone cyst.

Somebody probably wants me to talk about the syndrome characterized by osteitis fibrosa disseminata, areas of pigmentation and precocious puberty in females.* He was brown all over so he could not have brown spots. It is not that syndrome anyway. When that condition involves the shaft of a bone, it goes to the metaphysis and is not confined to the middle of the bone. Someone might consider xanthoma. I have seen the disease in a case associated with periosteal proliferation, but in the absence of other bone involvement it is most unlikely.

Because of the fever and elevated white count I am going to stick to the diagnosis of chronic pyogenic osteomyelitis. My second choice is bone cyst.

Dr. Tracy B. Mallory: Dr. Albright's diagnosis agrees with that of the local practitioner who sent the patient in, but its correctness was questioned for a long period of time in the hospital and many questions were raised. Have you anything to add, Dr. Hampton?

Dr. Hampton: I am startled with the simplicity of the case. We had several conferences over this patient, one with the Tumor Clinic. We started out with the impression that, by x-ray, the lesion in the bone was more like that of syphilis than of any other disease. His clinical manifestations, however, were not typical, and then we thought perhaps it was tuberculosis of the shaft which does occur in Negroes, Chinese and certain Scotchmen. However, tuberculosis usually involves the ends of the bone instead of the midshaft. We ended up by believing the lesion was due to an infection. One member thought that a Ewing tumor was a definite possibility. It was not so simple to us as it was to Dr. Albright.

Dr. Grantley W. Taylor: We had the problem of disposing of this patient and did not know in which of several directions to send him. He was on the osteomyelitis service, but Dr. Daland had the bone-tumor assignment, and the syphilologists began to "lick their chops" as soon as the positive blood test was returned. It seemed simplest to let the syphilologists try intensive antisyphilitic treatment. So this was carried out for a period of two weeks, without any striking improvement. Against the diagnosis of syphilis was the complete

*Albright, F.; Butler, A. M.; Hampton, A. O., and Smith, P.: Syndrome characterized by osteitis fibrosa disseminata, areas of pigmentation and endocrine dysfunction, with precocious puberty in females. *New Eng. J. Med.* 216:727-746, 1937.

freedom from other evidence of the disease. The arm symptoms had started six years before entry, at the age of nineteen, and hence if the process were due to primary syphilis, the disease must have been acquired at an earlier time; however, there was no history, and no other stigmas. We wondered if this might have been a false-positive Hinton test, and a syphilologist raised the question as to whether the febrile reaction might have accounted for the positive test. It was a very interesting possibility which I had not seen brought out before.

We tried aspiration with a needle and got nothing. Because of that we finally explored him. We got a copious immediate discharge of pus, and when a probe was introduced the length of the cavity, more pus was evacuated. Cultures showed *Staphylococcus aureus*. He went ahead to a very rapid, serene convalescence. He was discharged after two weeks, with no symptoms—a most bizarre convalescence for a case of osteomyelitis. Tissue was given to the Pathological Department, and I believe Dr. Mallory will tell us that it showed a distinctly benign sort of process for osteomyelitis.

DR. HAMPTON: What were the gross findings in the shaft?

DR. TAYLOR: There was a thickened, edematous, red, porky periosteum, with practically no cortex. We dropped immediately into an abscess cavity, and inside of that was the usual chronic inflammatory, granulation tissue.

CLINICAL DIAGNOSIS

Tertiary lues?
Osteomyelitis?

DR. ALBRIGHT'S DIAGNOSIS

Chronic pyogenic osteomyelitis.

ANATOMICAL DIAGNOSIS

Chronic osteomyelitis of humerus, *Staphylococcus aureus*.

PATHOLOGICAL DISCUSSION

DR. MALLORY: The clinical course, I think, is the most important proof that the *Staphylococcus aureus* was at least the immediate cause of this patient's difficulties, because as soon as the abscess was drained, as Dr. Taylor has pointed out, he had an immediate symptomatic improvement, which continued on to apparently complete cure. The material excised from the margins of the cavity appears to consist of granulation tissue, very heavily infiltrated with lymphocytes and plasma cells; there are quite a lot of lipoid-filled cells, but not enough to make one think seriously of xanthoma. I think there is a possibility that something existed before the abscess developed, but I do not know any way of proving it.

DR. HAMPTON: This is not the first time our department has had considerable difficulty with a staphylococcal abscess. We have quite a series of them. They mimic bone tumors and practically everything else, especially when the lesion has been present a long time.

DR. ERNEST M. DALAND: You cannot find any evidence of syphilis?

DR. MALLORY: No. I should think if syphilis entered the case at all it was congenital rather than acquired.

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ANTI-VIVISECTION

IN an attractive illustrated folder the people of New England are being asked by the New England Anti-Vivisection Society to support the "cause of anti-vivisection" as being "the cause of all right-thinking individuals." Since most of us secretly pride ourselves on our right thinking we believe that this appeal is addressed to us.

We are told that thousands of people are learning for the first time that vivisection really means animal experimentation, that the "path of cruelty" does not lead to man's birthright, nor will man find relief from pain in the "broken and tortured bodies of those weaker than themselves" and that among the victims of vivisection are not only animals but also "the less fortunate among human beings." A police dog looks out from the page with eyes of canine honesty. A kitten appears, to typify helplessness. The human victims do not appear.

This folder purports to be a sincere appeal to the people of New England to correct an evil by the abolishment of animal experimentation, a procedure which is motivated by cruelty and exists for the purpose of causing suffering to animals and unfortunate human beings. Need any intelligent person be disturbed by such an appeal?

The fight against cruelty in all its forms has always appealed and will always appeal to the best in human nature—of which, be it said in passing, the medical profession has its full share. Experimentation on living creatures is essential to man's knowledge of disease, on which in turn is based a large amount of what progress has been made against suffering and pain not only in human beings but also in animals. Should the misguided appeal of the New England Anti-Vivisection Society fail to achieve the universal support of the people to whom it is addressed, such failure cannot fairly be credited to any lack of right thinking in New England.

SOURCES OF COMMON MEDICAL EPONYMS

THE most graphic and essentially the best description of a clinical syndrome or a medical procedure is commonly that given by its discoverer or originator in the first flush of his enthusiasm. He may not have known so much about the matter as hundreds of his successors, but what he said was the kernel about which subsequent knowledge grew. To give him the tribute of an eponym seems quite fitting and proper, and not unscientific, and hundreds of names of physicians and scientists are associated with various diseases, syndromes, signs, tests, anatomic parts, pathologic lesions and so forth. Such terms as Bright's disease, Horner's syndrome, Hegar's sign, Barany's test, McBurney's point and Brodie's abscess are constantly used by the medical profession, but few physicians have read the original contributions by Bright, Horner, Hegar, Barany, McBurney and Brodie in regard to these matters.

Some time ago, Dr. Robert W. Buck, of Boston, submitted to the *Journal* a series of several hundred sources of common medical eponyms

which he had collected over a period of years, and beginning with this issue, one will be published weekly. While the list selected by the editorial staff is purely arbitrary and makes no pretense of being encyclopedic, it is believed that the individual items are of sufficient general interest to constitute a valuable addition to the *Journal*.

MEDICAL EPONYM

Adams-Stokes Disease

Robert Adams (1791-1875), one of the surgeons to Jervis Street Infirmary, Dublin, describes "Cases of Diseases of the Heart, Accompanied with Pathological Observations" in the *Dublin Hospital Reports* (4:353-453, 1827). On page 391 appears the following:

> February 20, 1822, I was called to visit a gentleman in my neighbourhood, aged 50 years, who had suddenly fallen down, as reported to me, in an apoplectic fit. I found him in a state of complete insensibility; his face (naturally pale and sickly) was now red and bloated; his breathing stertorous, with a slow pulse. . . . In the last year he had two apoplectic attacks, exactly resembling that which I had just witnessed: from these he recovered without any paralysis of the muscles. . . .
>
> An officer in the revenue, aged 68 years, of a full habit of body, had for a long time been incapable of any exertion, as he was subject to oppression of his breathing and continued cough. In May 1819 . . . I saw this gentleman: he was just then recovering from the affects of an apoplectic attack, which had suddenly seized him three days before. . . . What most attracted my attention was the irregularity of his breathing, and remarkable slowness of the pulse, which generally ranged at the rate of 30 in a minute. . . . [During seven years he had been seen] in not less than twenty apoplectic attacks. . . . He would then fall down in a state of complete insensibility. . . . His pulse would become even slower than usual. . . . He recovered from these attacks without any paralysis. . . . In both these cases . . . apoplexy must be considered less a disease in itself than symptomatic of one, the organic seat of which was in the heart.

William Stokes (1804-1878) in his article "Observations on Some Cases of Permanently Slow Pulse," which appeared in the *Dublin Quarterly Journal of Medical Science* (2:73-85, 1846), refers to these case histories as follows:

> In the fourth volume of the *Dublin Hospital Reports*, Mr. Adams has recorded a case of permanently slow pulse, in which the patient suffered from repeated cerebral attacks of an apoplectic nature, though not followed by paralysis. The attention of subsequent writers on diseases of the heart has not been sufficiently directed to this case, which is an example of a very curious and, as there is reason to believe, special combination of symptoms.

R. W. B.

MASSACHUSETTS MEDICAL SOCIETY

SECTION OF OBSTETRICS AND GYNECOLOGY*

Raymond S. Titus, M.D., *Secretary*
330 Dartmouth Street
Boston

Puerperal Sepsis Following Delivery

Mrs. M. F., a thirty-year-old para IV, was delivered normally at home on July 19, 1926. There had been some difficulty in extracting the placenta. Following delivery the patient was weak and apathetic. On July 20 the patient complained of a sharp frontal headache and a pain in the back of her neck, which was worse on motion. She was therefore admitted to the hospital.

The family history was not recorded. The patient's past history was not remarkable. The three previous deliveries had been normal, with uneventful puerperiums.

Examination on entry revealed the temperature 102.4°., the pulse 140, and the respirations 24. The patient was conscious, rational and co-operative. The heart was not enlarged; there were no murmurs. The lungs were clear and resonant; there were no rales. The uterus was at the umbilicus; there was no tenderness, spasm or masses in the abdomen. The extremities were normal. The blood showed a white-cell count of 16,400, and the urine contained many pus cells.

She was given ice to the fundus, rectal fluids, urinary antiseptics, bladder irrigations and ergot. Improvement was gradual. For several days the temperature varied between 100 and 103°F., reaching a normal level on July 31. The white-cell count rose to 19,600 on July 26. The patient was discharged on August 8, completely recovered.

Comment. This is a typical case of uterine infection developing within thirty-six hours of delivery. The history states that some difficulty had been encountered in extracting the placenta. One does not know whether the placenta was extracted manually. If so, the infection was probably due to direct contamination. The patient's condition as described by the attending physician soon after delivery leads one to infer that hemorrhage was a complication. It is often true that patients whose temperatures rise within thirty-six hours are much sicker than those whose temperatures do not rise until after three days. No blood culture was taken. It is mentioned in the record that the patient was given ergot; it is not mentioned when the ergot

*A series of selected case histories by members of the section will be published weekly. Comments and questions by subscribers are solicited and will be discussed by members of the section.

was discontinued. It must always be borne in mind that continued use of ergot is dangerous and that its value after a few days is probably negligible.

MISCELLANY

RÉSUMÉ OF COMMUNICABLE DISEASES IN MASSACHUSETTS FOR NOVEMBER, 1939

DISEASES	NOVEMBER 1939	NOVEMBER 1938	FIVE-YEAR AVERAGE*
Anterior poliomyelitis	7	0	14
Chickenpox	950	812	984
Diphtheria	28	21	28
Dog bite	553	656	605
Dysentery, bacillary	135	9	11
German measles	36	30	62
Gonorrhea	426	421	514
Lobar pneumonia	269	233	260
Measles	857	597	384
Meningococcus meningitis	2	2	4
Mumps	248	257	347
Paratyphoid B fever	1	12	4
Scarlet fever	264	356	559
Syphilis	419	503	477
Tuberculosis, pulmonary	162	262	259
Tuberculosis, other forms	9	30	28
Typhoid fever	2	6	9
Undulant fever	5	5	3
Whooping cough	508	557	607

*Based on figures for preceding five years.

RARE DISEASES

Anterior poliomyelitis was reported from: Adams, 1; Groton, 1; Haverhill, 1; Worcester, 4; total, 7.

Anthrax was reported from: Peabody, 1; total, 1.

Diphtheria was reported from: Boston, 1; Cambridge, 4; Fall River, 3; Foxboro, 5; Lawrence, 5; Lynn, 1; Marlboro, 1; New Bedford, 1; North Andover, 1; Revere, 2; Taunton, 1; Worcester, 3; total, 28.

Dysentery, bacillary, was reported from: Arlington, 1; Beverly, 5; Boston, 1; Cambridge, 11; Chelmsford, 1; Danvers, 12; Fall River, 2; Lowell, 1; Lynn, 1; Malden, 5; Medfield, 69; Newton, 2; Palmer, 3; Russell, 1; Salem, 3; Waltham, 4; Watertown, 1; Westport, 3; Wrentham, 9; total, 135.

Malaria was reported from: Boston, 1; total, 1.

Meningococcus meningitis was reported from: Brockton, 1; Cambridge, 1; total, 2.

Paratyphoid B fever was reported from: Woburn, 1; total, 1.

Pellagra was reported from: Boston, 1; total, 1.

Pfeiffer bacillus meningitis was reported from: Wakefield, 1; Worcester, 2; total, 3.

Septic sore throat was reported from: Boston, 3; Brookline, 1; Cambridge, 1; Malden, 2; Merrimac, 1; Somerville, 1; Williamstown, 1; total, 10.

Trachoma was reported from: Boston, 2; Cambridge, 1; Worcester, 1; total, 4.

Trichinosis was reported from: Ayer, 1; Boston, 1; total, 2.

Typhoid fever was reported from: Cambridge, 1; Saugus, 1; total, 2.

Undulant fever was reported from: Beverly, 1; Dighton, 1; Gloucester, 1; Leverett, 1; North Adams, 1; total, 5.

Diphtheria for the eighth consecutive month showed a higher reported incidence than for the corresponding month of the previous year.

Lobar pneumonia, after four low months, showed an increase to slightly above the five-year average.

Sonne (bacillary) dysentery continues to be reported at an unusually high level. All parts of the state have been involved, with a concentration of reported cases in the Metropolitan Boston area.

Typhoid fever was reported at a record low level.

For the fifth consecutive month the reported incidence of scarlet fever reached a new low.

The reported incidences of pulmonary tuberculosis and tuberculosis (other forms) were well within the five-year average.

Measles continued in its seasonal upswing, reaching the highest reported November level for the previous five years.

There was nothing noteworthy about the reported incidences of anterior poliomyelitis, meningococcus meningitis, paratyphoid B fever, undulant fever, whooping cough, chickenpox, German measles and mumps.

REPORTS OF MEETINGS

BOSTON CITY HOSPITAL

On November 9, 1939, at the Boston City Hospital, Dr. A. C. Williams of the House Officers' Association presented Dr. George Crile, of Cleveland, who discussed "Hypertension, Can It Be Cured?" The importance of the problem is indicated, among other things, by the actuarial statistics of several large insurance companies, which show that fifty cents of each dollar paid in the past few years has been for deaths from hypertension and coronary and cardiorenal diseases.

Dr. Crile's unique approach to the subject resulted from his investigation of the explosion ten years before of some x-ray films which had been brought about by only a slight temperature rise. That led to a comparison of explosives, x-ray film, and protoplasm as regards composition. He concluded that the last was much like the other explosives qualitatively and was in a constant state of nitro-disintegration influenced largely by the thyroid and adrenal glands.

Dr. Crile then undertook a ten-year research into the reason for the susceptibility of the "civilized" person to certain degenerative diseases practically, if not entirely, unknown to native tribes and animals. Over thirty-five hundred animals were obtained and weighed in toto, and their various organs were then weighed individually. It was an attempt fundamentally to determine wherein lay the "intelligence, personality and energy of man," and attention was directed primarily at the brain, sympathetic nervous system, adrenal glands and thyroid gland. The speaker had pictures demonstrating the gradual increase in size of these organs through the phylogenetic scale, with a great step forward being noted between the cold- and warm-blooded animals. This was not considered surprising, since it has been estimated that 75 per cent of the daily food intake is used merely for the maintenance of the warm-blooded state. By far the largest celiac plexus was found in the lion and tiger, which have developed this in response to their need for power and alertness for the "supreme effort" which characterizes their survival. Dr. Crile concluded from his exhaustive study that the size of the brain is proportional to the environmental temperature, its greater part being used for "sparking the body metabolism."

Although man did not head the list in regard to size of the celiac plexus, the ratios of the weights of the brain, thyroid gland and adrenal glands to the body weight showed him far in the lead. This, Dr. Crile believes, is the reason man is the world's most energized being and is one of the reasons for man's place in nature. Another factor is his unique ability to control his body heat by virtue of his great body surface, lack of natural body covering and unique faculty for sweating all over.

Furthermore, man was the only animal in whom the ratio of thyroid weight to the total body weight surpassed the adrenal ratio. Consequently he is not endowed with the tremendous capacity for energy outbursts of some of his animal counterparts but is instead a walking, thinking being with the singular and often undesirable tendency to work all day and worry all night.

Then Dr. Crile attempted to determine whether animals obeyed any physical laws in regard to their metabolism. The only correlation following any such law was between the brain weight and the amount of energy produced in twenty-four hours. Man, and to a lesser degree the primates, were the only exceptions to the law; and since Benedict ha.. ruled out mental processes as contributing any significant amount to human metabolism, this was taken as another indication that man really has a unique place in nature. The speaker considered this somewhat of an "anatomical I. Q." for man. The only drive peculiar to man is his thinking brain, and this may be the force, through the thyroid and adrenal glands, which causes diseases specific for civilized man.

The next step was the correlation of this natural history with the problems of cardiovascular disease which had prompted the extensive research. Dr. Crile corroborated the well-known fact that the larger size of the brain in civilized man is due to its increase in the "thinking portion" of the organ over that of the aborigine. However, there was a concomitant increase of the thyroid and adrenal glands, and it was the size of the latter and the celiac plexus which attracted the attention of Dr. Crile. Consequently, the weights of the ganglia were compared in known hypertensive states and in various other conditions. The preponderance of greater weights in the former disease was marked. This, then, concluded the speaker, was a point of attack on hypertension, an opportunity to correct the pathologic physiology by interruption of the anatomical pathway.

A total of 256 patients have been subjected to celiac ganglionectomy. Particular attention was focused on their early lives. It was found that their childhood differed from that of eventual thyrotoxic patients but that both were far different from normal children. Both groups were found to have been high-tension, tireless children with many activities and ambitions. Those who later showed hyperthyroid tendencies, however, were, on the average, far superior academically. In both groups the people themselves seldom became fatigued but the integral parts wore out.

At first, the number of operative and postoperative fatalities made the success of the procedure questionable. With additional experience, however, Dr. Crile and his associates have completed the past 127 cases without an operative death. The early misfortunes resulted from the technical difficulties of the procedure itself, the fall of the blood pressure to zero in some instances, and the permanent anuria from long-continued hypotension. To combat these complications, it was decided to give continuous intravenous glucose in distilled water, and perform the operation slowly and with meticulous care.

The average results three years postoperatively proved promising, stated Dr. Crile, for the average blood pressure had fallen from 213 systolic, 130 diastolic, to 187, 116. The fine results in many instances, however, were neutralized by the failures in improperly selected cases. At that time there were no adequate criteria for operability, even those who had had apoplectic attacks being included. In spite of that, however, 55 per cent survived three years or more and 82 per cent returned to work, many in spite of lack of objective improvement. There was symptomatic relief in 88 per cent of cases, and this, Dr. Crile opined, was due to a damping of the patient's "innate" drive, in spite of the progression of the already existing arteriosclerosis in many patients. In fact, it was doubtful in such cases if there was any prolongation of life. But just the relief from symptoms, which the speaker said he had come to realize were worse than those ascribed to terminal cancer, was considered a justification for the ganglionectomy. The decrease in cardiac deaths from 60 to 12 per cent was considered an indication for further study in the same direction.

Dr. Crile's final suggestion was for an attempt at the prevention of hypertension by recognizing the children possessing the innate tendencies. These children should not be allowed to skip grades at school and engage in outside activities but should rather be urged to enjoy life in a wholesome way. The speaker stated that satisfaction of ambitions merely allows the inborn driving force within the brain to cause hyperfunction of the sympathicoadrenal system, with resultant hypertension. It was pointed out that those practicing the Chinese philosophy in its classic form have practically no hypertension as compared with their countrymen in Hong Kong.

In discussion, Dr. A. R. Kimpton asked if renal omentopexy might not be beneficial following the operation, in order to help the already damaged kidney. Dr. Crile replied that such was an excellent idea.

A physician asked whether these patients lost their entire energy. But Dr. Crile assured him that such was not the case, citing the instance of a successful surgeon who was still carrying on, albeit somewhat more slowly, after the removal of the largest celiac ganglion ever weighed. He said that there is a subjective loss of a certain drive, but the presence of a large initial amount makes the final result entirely acceptable.

NOTICES

BOSTON CITY HOSPITAL

The monthly clinicopathological conference will be held at the Boston City Hospital on Wednesday, January 10, at 12 o'clock noon, in the Pathological Amphitheater.

JOSEPH E. HALLISEY, M.D., *Secretary*,
Medical Staff.

SOUTH END MEDICAL CLUB

The next meeting of the South End Medical Club will be held at the headquarters of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston, on Tuesday, January 16, at 12 o'clock noon. Dr. Siegfried J. Thannhauser will speak on "Obesity."

Physicians are cordially invited to attend.

JOHN B. HALL, M.D., *Secretary*.

PETER BENT BRIGHAM HOSPITAL

A research conference of the medical staff of the Peter Bent Brigham Hospital will be held in the amphitheater of the hospital on Tuesday, January 9, at 5 p.m.

PROGRAM

The Observations of Diuresis Produced by Hypertonic Sugar and Polyhydric Alcoholic Solutions. Dr. Philip Grabfield.

Toxemia of Pregnancy. Dr. Lewis Dexter.

A joint medical and surgical clinic at the Peter Bent Brigham Hospital will be held on Wednesday, January 10,

from 2 to 4 p.m. Drs. Soma Weiss and Elliott C. Cutler will speak on "Chills." A clinicopathological conference conducted by Dr. Elliott C. Cutler will take place from 4 to 5 p.m.

On Thursday, January 11, from 8:30 to 9:30 a.m. there will be at the Peter Bent Brigham Hospital, a combined clinic, conducted by Dr. Soma Weiss, of the medical, surgical, orthopedic and pediatric services of the Children's Hospital and the Peter Bent Brigham Hospital.

Physicians and students are cordially invited to attend.

TUMOR CLINIC, BOSTON DISPENSARY

Each Tuesday and Friday morning, 10:00 to 12:30, there is a meeting of the Tumor Clinic of the Boston Dispensary, a unit of the New England Medical Center. Neoplasms of various sorts are seen and discussed, and when there is an indication, are treated with radium of high-voltage x-ray. Physicians are invited to visit this clinic. They may bring patients for aid in diagnosis or may refer patients to the clinic following which a report will be returned to the referring physician. A limited number of beds are available for diagnostic study and for treatment.

BOSTON DOCTORS' SYMPHONY ORCHESTRA

The Boston Doctors' Symphony Orchestra will rehearse under Alexander Theide, former concert-master with the Cleveland Symphony Orchestra and the Philadelphia Symphony Orchestra, every Thursday at 8:30 p.m., in Studio A, Station WMEX, 70 Brookline Avenue, Boston. Those interested in becoming members should communicate with Dr. Julius Loman, Pelham Hall Hotel, Brookline (BEA 2430).

HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held on Tuesday, January 9, in the amphitheater of the Peter Bent Brigham Hospital (Shattuck Street entrance), at 8:15 p.m.

PROGRAM

Presentation of cases.

The Mechanism of Secretion in the Adrenal Gland. Dr. H. Stanley Bennett.

Medical students and physicians are cordially invited to attend.

ROBERT M. ZOLLINGER, M.D., *Secretary.*

UNITED STATES MARINE HOSPITAL

The staff meeting of the United States Marine Hospital, Chelsea, Massachusetts, will be held at "The Hut," on Friday afternoon, January 19, at 4:00. Dr. H. Houston Merritt will talk, his subject being "Syphilis of the Central Nervous System."

JOHN W. TRASK, *Medical Director in Charge.*

SECOND ANNUAL CONGRESS ON INDUSTRIAL HEALTH

The Second Annual Congress on Industrial Health, sponsored by the Council on Industrial Health of the American Medical Association, will be held at the Palmer House, Chicago, on Monday and Tuesday, January 15 and 16.

The congress will have a morning and afternoon session devoted to special topics each day. "Syphilis in Industry" will be discussed at the Monday afternoon session, "Physical Examination" on Tuesday morning and "Disability Evaluation" on Tuesday afternoon.

A dinner and round-table discussion on "Organization and Activities of Committees on Industrial Health in State and County Medical Societies" will take place on the first evening, January 15.

SOCIETY MEETINGS AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, JANUARY 8

MONDAY, JANUARY 8

*12:15 p.m.–1:15 p.m. Clinicopathological conference. Dr. S. Burt Wolbach, Peter Bent Brigham Hospital amphitheater.

*4 p.m. Physicians and medical students are cordially invited to attend a clinic presented by the medical, surgical and orthopedic services of the Infants' and Children's hospitals in the amphitheater of the Children's Hospital.

TUESDAY, JANUARY 9

*9–10 a.m. Certain Hematological Problems. Dr. W. Dameshek. Joseph H. Pratt Diagnostic Hospital.

*10 a.m.–12:30 p.m. Boston Dispensary tumor clinic.

*12:15 p.m.–1:15 p.m. X-ray conference. Dr. Merrill C. Sosman. Peter Bent Brigham Hospital amphitheater.

5 p.m. Research conference of the medical staff. Peter Bent Brigham Hospital.

*8:15 p.m. Harvard Medical Society. Peter Bent Brigham Hospital (Shattuck Street entrance).

WEDNESDAY, JANUARY 10

*9–10 a.m. Hospital case presentation. Dr. S. J. Thannhauser. Joseph H. Pratt Diagnostic Hospital.

*12 m. Clinicopathological conference. Children's Hospital amphitheater.

12 m. Monthly clinicopathological conference. Pathological amphitheater, Boston City Hospital.

*2 p.m.–4 p.m. Joint medical and surgical clinic. Peter Bent Brigham Hospital.

THURSDAY, JANUARY 11

*8:30 a.m.–9:30 a.m. Combined clinic of the medical, surgical, orthopedic and pediatric services of the Children's Hospital and the Peter Bent Brigham Hospital, at the Peter Bent Brigham Hospital.

*9–10 a.m. The Place of Electrocardiography in Clinical Diagnosis. Dr. J. M. Faulkner. Joseph H. Pratt Diagnostic Hospital.

FRIDAY, JANUARY 12

*9–10 a.m. The Present-Day Specific Treatment of Pneumonia. Dr. Maxwell Finland. Joseph H. Pratt Diagnostic Hospital.

*10 a.m.–12:30 p.m. Boston Dispensary tumor clinic.

SATURDAY, JANUARY 13

*9–10 a.m. Hospital case presentation. Dr. S. J. Thannhauser. Joseph H. Pratt Diagnostic Hospital.

*10 a.m.–12 m. Medical staff rounds of the Peter Bent Brigham Hospital. Conducted by Dr. Soma Weiss.

SUNDAY, JANUARY 14

4 p.m. Serious Accidents: What to do and what not to do. Dr. Charles C. Lund. Free public lecture. Harvard Medical School, amphitheater of Building D.

*Open to the medical profession.

JANUARY 5 — United States Marine Hospital. Page 1001, issue of December 21.

JANUARY 6, JUNE 8–11 — American Board of Obstetrics and Gynecology. Page 160, issue of July 27, and page 798, issue of November 16.

JANUARY 9 — Research conference of the medical staff. Peter Bent Brigham Hospital. Page 38.

JANUARY 9 — Harvard Medical Society. Notice above.

JANUARY 10 — Joint medical and surgical clinic. Peter Bent Brigham Hospital. Page 38.

JANUARY 10 — Monthly clinicopathological conference. Boston City Hospital. Page 38.

JANUARY 11 — Combined clinic of the medical, surgical, orthopedic and pediatric services of the Children's Hospital and the Peter Bent Brigham Hospital. Page 38.

JANUARY 11 — Pentucket Association of Physicians. 8:30 p.m., Hotel Bartlett, Haverhill.

JANUARY 12 — United States Marine Hospital. Page 1043, issue of December 28.

JANUARY 14 — Harvard Medical School, free public lecture. Page 1042, issue of December 28.

JANUARY 14 — Salem Hospital public lecture. Page 1042, issue of December 28.

JANUARY 15 and 16 — Second Annual Congress on Industrial Health. Page 39.

JANUARY 15 and 22 — Cutter Lectures. Page 1042, issue of December 28.

JANUARY 16 — South End Medical Club. Page 38.

JANUARY 19 — United States Marine Hospital, staff meeting. Page 39.

JANUARY 22–25 — American Academy of Orthopaedic Surgeons. Hotel Statler, Boston.

FEBRUARY 11–14 — International College of Surgeons. Page 759, issue of November 9.

FEBRUARY 22–24 — American Orthopsychiatric Association. Page 957, issue of December 14.

MARCH 2, JUNE 8 and 10 — American Board of Ophthalmology. Page 719, issue of November 2.

MARCH 7–9 — The New England Hospital Association. Hotel Statler, Boston.

MAY 10–18 — American Scientific Congress. Page 1043, issue of December 28.

MAY 14 — Pharmacopoeial Convention. Page 894, issue of May 25.

JUNE 7–9 — American Board of Obstetrics and Gynecology. Page 1019, issue of June 15.

District Medical Societies

ESSEX SOUTH

FEBRUARY 14 — Cough, Sputum, Hemoptysis — How shall they be investigated? Dr. Reeve H. Betts. Essex Sanatorium, Middleton.

MARCH 6 — Experimental and Clinical Considerations of Sulfanilamide Treatment of Hemolytic Streptococcal Infections. Dr. Champ Lyons. Lynn Hospital, Lynn.

APRIL 3 — Addison Gilbert Hospital, Gloucester.

MAY 8 — Annual meeting. Salem Country Club, Peabody.

HAMPSHIRE

JANUARY 10.

MARCH 13.

MAY 8.

Meetings are held at 11:30 a.m. at the Cooley Dickinson Hospital, Northampton.

MIDDLESEX EAST

JANUARY 10.

MARCH 20.

MAY 15.

Meetings are held at 12:15 p.m. at the Unicorn Country Club, Stoneham.

MIDDLESEX NORTH

JANUARY 31.

APRIL 24.

JULY 31.

OCTOBER 30.

NORFOLK SOUTH

FEBRUARY 1.

MARCH 7.

APRIL 4.

MAY 2.

All meetings, with the exception of one which is usually held at the Quincy City Hospital, are held at the Norfolk County Hospital in South Braintree, at 12 o'clock noon.

PLYMOUTH

JANUARY 18 — Brockton Hospital, Brockton.

MARCH 21 — Goddard Hospital, Brockton.

APRIL 18 — State Farm.

MAY 16 — Lakeville Sanatorium, Lakeville.

SUFFOLK

JANUARY 31 — Scientific meeting. Subject to be announced later.

MARCH 27 — Scientific meeting. Symposium on Ulcerative Colitis and Diarrheas. Under the direction of Dr. Chester M. Jones.

APRIL 24 — Annual meeting in conjunction with the Boston Medical Library. Election of officers. Program and speakers to be announced later.

WORCESTER

JANUARY 10 — Worcester City Hospital.

FEBRUARY 14 — Worcester State Hospital.

MARCH 13 — Worcester Memorial Hospital.

APRIL 10 — Worcester Hahnemann Hospital.

MAY 8 — Worcester Country Club.

Each meeting begins with a dinner at 6:30 p.m. and is followed by a business and scientific meeting.

BOOK REVIEWS

Functional Disorders of the Foot: Their diagnosis and treatment. Frank D. Dickson and Rex L. Diveley. 305 pp. Philadelphia, Montreal and London: J. B. Lippincott Co., 1939. $5.00.

The authors of this text very justly point out that the prevalence and economic importance of foot disorders are not sufficiently appreciated by the medical profession; that the profession treats them too indifferently; and that a large portion of the public has grown to consider these ailments as lying more directly in the province of such lay groups as chiropractors and shoe salesmen rather than in that of the family doctor. Unfortunately, there is altogether too much truth in these assertions. To help remedy the situation, this text is aimed principally as a guide for the practicing physician toward a clearer understanding of common foot disorders. Secondarily, the authors seek to offer the non-medical practitioners an exposition of "the primary causes of foot disorders and their legitimate limitations in carrying out treatment." With this in view, it is explained that "only such material on the evolutionary development of the foot, on anatomy and on physiology has been introduced as seemed necessary to an intelligent understanding of the foot as a functioning organ. . . ." The aim which is thus expressed has been carried out most successfully, without limiting the completeness of the study.

The general causes of foot imbalance are first analyzed, and then succeeding chapters are devoted to the special influence of the three age groups — children, adolescents and adults. As the etiologic factors are considered, their clinical applications are described in sections devoted to symptoms, exercises and treatment. A special chapter deals with "foot apparel," another to foot strapping, and one to examination of the foot. There are also chapters discussing the common affections of the foot in the skin, nails, heel and hallux.

In general, the great value of this book lies in the intelligent evaluation which it places on various methods of treatment. It does not adopt any iron-clad radical or conservative rule, but reviews the various available corrective measures and selects that which is best suited to the particular type of case, whether by shoe correction, manipulation, stretching or operative procedure.

Studies from the Rockefeller Institute for Medical Research. Vol. 112. 600 pp. New York: The Rockefeller Institute for Medical Research, 1939. $2.00.

Of special interest in this volume is the study by Rous and Kidd comparing the virus-induced rabbit tumors with the tumors induced by the application of coal tar. There is a striking similarity between both types of papilloma.

An interesting and carefully studied case of periarteritis nodosa is presented by Emerson and others. A study by White brings out the potentially unlimited growth of excised plant callus under artificial conditions.

The Story of a Baby. Marie H. Ets. 63 pp. New York: The Viking Press, 1939. $2.50.

The author tells the story of a baby in a manner which will appeal to parents who wish enlightenment on a problem of this sort. It may also be read with much interest by high-school students and by laymen in general.